Exhibit 202

Opinions of Lewis A. Chodosh, M.D., Ph.D.

This report is offered pursuant to Rule 26 of the Federal Rules of Civil Procedure. Each of the opinions I have offered in this report is given to a reasonable degree of scientific and medical certainty, and is based on the methods and procedures of science, materials and literature I have reviewed in connection with this litigation, my knowledge of recognized medical and scientific principles, and methodology reasonably relied upon by members of my profession, as well as my education, training, knowledge, and experience.

My curriculum vitae is attached as Exhibit A to this report. During the previous 4 years, I have testified as an expert at 2 depositions (including my deposition in this litigation), and I have not testified as an expert in any trials. My fees charged in connection with this engagement are consistent with my normal practice for such work. My rate for reviewing materials, preparing reports, and deposition and trial testimony is $925 per hour.

A list of materials that I considered in rendering the opinions offered in this report is attached as Exhibit B. I reserve the right to supplement this list, as well as to amend and supplement the opinions expressed in this report. I also reserve the right to respond to and rebut all information provided in discovery, which I understand is ongoing, and any opinions offered by Plaintiffs' experts at their depositions or at trial.

I have been asked to provide my opinions regarding the scientific and medical basis for the requests for medical monitoring for cancer, as expressed in Plaintiffs' Third Amended Medical Monitoring Class Action Complaint filed November 1, 2021. As detailed in this Supplemental Report, my opinions regarding Plaintiffs' request for medical monitoring for cancer follow in a straightforward manner from the opinions expressed in my report of August 2, 2021 regarding the question of whether ingestion of valsartan products affects the risk of developing cancer, which is attached as Exhibit C. Where applicable, my response to each element of Plaintiffs' Third Amended Medical Monitoring Class Action Complaint refers to the relevant sections of my report of August 2, 2021.

Some citations to specific reference material are also offered in this Supplemental Report, where I believed it necessary to cite a specific source; otherwise, my opinions are derived from a combination of reference sources, my own scientific and clinical experience, general medical and scientific knowledge, and my report of August 2, 2021. This Supplemental Report is not intended to be an exhaustive recitation of all of my opinions.

A. Background and Qualifications

As summarized in my report of August 2, 2021 ¶¶ 1-15.

B. Summary of Plaintiffs' Claims Regarding Medical Monitoring

1. The Medical Monitoring Independent Claim Class and Medical Monitoring Remedy Class consist of Plaintiffs "*who consumed a sufficiently high Lifetime Cumulative Threshold of NDMA, NDEA, or other nitrosamine, in generic valsartan-containing drugs manufactured by or for Defendants and marketed in the United States and its territories and possessions, at least since Jan 1, 2012.*" Third Amended Medical Monitoring Class Action Complaint ¶¶ 538-539.

2. Plaintiffs indicate that they "*will use common evidence, including the use of a Lifetime Cumulative Threshold, to establish an increased risk of cancer classwide"*, which they define as a "*significantly increased risk of contracting* [cancer] *relative to what would be the case in the absence of exposure*". Memorandum of Law in Support of Plaintiffs' Motion for Class Certification § V.A.4.

3. Plaintiffs claim that they have "*defined both Classes with reference to objective criteria. The determination of whether the Class Member consumed a Lifetime Cumulative Threshold sufficient for Class membership is based on dosage, API manufacturer and time period*." Plaintiffs further claim that "*this information can be determined by examining pharmacy and prescription records, and by reference to the unique 10-digit NDC code issued to all generic drug products.*" Memorandum of Law in Support of Plaintiffs' Motion for Class Certification § III.B.

4. Specifically, Plaintiffs propose that "*the determination of whether the class member consumed a Lifetime Cumulative Threshold sufficient for class membership is based on objective and ascertainable factors*. *Specifically, (A) at a dose of 320 mg, the class member needs to have taken a combination of three (3) months of ZHP API, OR 18 months of Hetero API, OR 54 months of Mylan and/or Aurobindo API; (B) at a dose of 160 mg, the class member needs to have taken a combination of six (6) months of ZHP API, OR 32 months of Hetero API, OR 108 months of Mylan and/or Aurobindo API; (C) at a dose of 80 mg, the class member needs to have taken a combination of 12 months of ZHP API, OR 64 months of Hetero API, OR 216 months of Mylan and/or Aurobindo API; and (D) at a dose of 40 mg, the class member needs to have taken a combination of 24 months of ZHP API, OR 128 months of Hetero API, OR 432 months of Mylan and/or Aurobindo AP[.]*" Third Amended Medical Monitoring Class Action Complaint ¶¶ 540-541.

5. For each Plaintiff discussed, it is claimed that "*Plaintiff X consumed a Cumulative Lifetime Threshold of at least XXX units"* [of NDMA, NDEA, or other nitrosamine] and, as a result, "*suffered cellular and genetic injury that creates and/or increases the risk that Plaintiff will develop cancer.*" Third Amended Medical Monitoring Class Action Complaint ¶¶ 12-28.

6. Specifically, Plaintiffs claim elevated risks for developing esophageal, stomach, colorectal/intestinal, liver, lung, bladder, blood, pancreatic, and prostate cancers for those Plaintiffs who consumed valsartan products in amounts greater than the above Lifetime Cumulative Thresholds, and that such Plaintiffs "*require additional targeted testing*" consisting of periodic fecal occult blood testing, low does CT chest scan, urinalysis, blood

smear evaluation, colonoscopy and upper endoscopy, and Galleri multi-cancer early detection blood test or similar liquid biopsy. Memorandum of Law in Support of Plaintiffs' Motion for Class Certification § II.B.3.

7. Plaintiffs claim that reports from Drs. Madigan and Panigrahy "*demonstrate, using dietary and other studies, that there is a threshold NDMA Lifetime Cumulative Exposure associated with statistically significant increased risks of developing cancers.*" Plaintiffs also claim that *"Specifically, Dr. Madigan analyzed dietary and other studies to demonstrate the Lifetime Cumulative Thresholds of NDMA that trigger statistically significant increased risks for different types of cancers.*" Memorandum of Law in Support of Plaintiffs' Motion for Class Certification §§ II.B.2, V.A.4.

8. In his report, Dr. Panigrahy refers to lifetime cumulative dietary exposures to NDMA that were associated with statistically significant increased risks of gastric (5,913 ug, 4,161 ug), colorectal (1,160 ug, 4,161 ug, 2,759 ug) and lung cancer (15,330 ug) in some published dietary epidemiology studies. However, he notes that these "*should not be considered as bright line thresholds*". Report of Dr. Dipak Panigrahy July 6, 2021 at 90.

9. In his report, Dr. Madigan relies upon dietary epidemiology studies to derive Lifetime Cumulative Exposures to NDMA that he considers to be associated with a statistically significant increased risk of specific cancers. Specifically, Dr. Madigan highlights Lifetime Cumulative Exposures to NDMA associated with increased risks of cancer as low as 1,962 ug (gastric cancer), 3,343 ug (rectal cancer), 4,235 ug (esophageal cancer) and 4,303 ug (lung cancer), as well as Lifetime Cumulative Exposure to NDEA associated with increased risks of cancer as low as 2,520 ug for pancreas cancer. The dietary epidemiology studies Dr. Madigan relies upon failed to identify statistically significant increased risks of cancer associated with NDMA for bladder cancer, prostate cancer, pancreas cancer, liver cancer, or blood cancers. Report of Dr. David Madigan July 7, 2021 ¶¶ 23-26, 33, Table 1.

10. Dr. Madigan also concludes based upon the occupational exposure study of Hidajat et al. that "*cumulative exposure to greater than 7,514 ug of NDMA[27] statistically significantly increases one's risk of developing the following cancers – bladder, lung, stomach, multiple myeloma, esophageal, prostate, and prostate. Cumulative exposure greater than 14,319 ug of NDMA adds leukemia, lymphoma, and liver to the list.*" Report of Dr. David Madigan July 7, 2021 ¶ 34.

C. Opinions

11. For the reasons outlined below and discussed in detail in my report of August 2, 2021, Plaintiffs' claim that, for example, ingestion of valsartan 320 mg tablets of ZHP API for 3 months, OR Hetero API for 18 months, OR Mylan and/or Aurobindo API for 54 months, is associated with elevated risks for developing esophageal, stomach, colorectal/intestinal, liver, lung, bladder, blood, pancreatic, and prostate cancers, is not supported by medical or scientific evidence, is contradicted by a wealth of medical and scientific evidence, is

internally inconsistent, and lacks a coherent logical basis. Moreover, Plaintiffs' selection of a specific Lifetime Cumulative Exposure threshold to trigger lifetime medical monitoring for cancer is arbitrary, subjective and lacks a reliable medical or scientific basis.

12. Indeed, even the durations of exposure to valsartan products potentially containing NDMA or NDEA that are proposed by Plaintiffs' algorithm to be sufficient to trigger lifetime medical monitoring (*e.g.*, 108 months, 128 months, 216 months, or 432 months) are nonsensical and untethered to reality, given that the maximum period of time that valsartan products potentially containing NDMA were available on the U.S. market was only 51 months (9/29/2014-12/31/2018), and that the maximum period of time that valsartan products potentially containing NDEA were available on the U.S. market was only 75 months (9/21/2012-12/31/2018). This fact alone highlights the lack of correspondence between Plaintiffs' proposed algorithm for determining Lifetime Cumulative Exposure thresholds and any reasonable – or even vaguely logical – understanding of the facts regarding potential exposures to NDMA and/or NDEA attributable to ingestion of valsartan products that were even physically possible.

Plaintiffs' proposed approach cannot ascertain actual NDMA or NDEA exposures

13. Plaintiffs have failed to articulate a logical medical or scientific basis for their claimed dose/duration/API thresholds for medical monitoring, as no basis for these thresholds is stated either in the Third Amended Medical Monitoring Class Action Complaint or in the Memorandum of Law in Support of Plaintiffs' Motion for Class Certification. Moreover, the dose/duration/API thresholds proposed by Plaintiffs cannot be used to determine actual NDMA or NDEA exposures for individual Plaintiffs.

14. Plaintiffs claim that NDMA and NDEA exposures can be established for each Plaintiff based upon the dosage of valsartan product that they ingested, the manufacturer of the API, and the "*time period*" as "*determined by examining pharmacy and prescription records, and by reference to the unique 10-digit NDC code issued to all generic drug products.*" Memorandum of Law in Support of Plaintiffs' Motion for Class Certification § III.B. From context, "*time period*" appears to refer to duration of exposure to valsartan products.

15. In contrast to Plaintiffs' claim, this approach could not conceivably establish the NDMA and/or NDEA level to which any given Plaintiff may have been exposed. That is, knowledge of the dosage of drug ingested by a Plaintiff, the API manufacturer, and the duration of drug treatment does not specify a level of NDMA or NDEA exposure. This follows from the fact that the NDMA and NDEA content of different valsartan products varied widely across different manufactured lots of valsartan API, in part as a function of calendar date, even from the same manufacturer. Many of these lots of valsartan API contained levels of NDMA and/or NDEA that were either below the limits of detection or below the FDA threshold of Acceptable Daily Intake (ADI). Since the 10-digit National Drug Code (NDC) on generic products upon which Plaintiffs propose to rely does not specify the manufactured lot of API, it is clear that the algorithm proposed by Plaintiffs cannot be used to determine a specific exposure to NDMA or NDEA. Consequently, it cannot possibly be the case that Plaintiffs' proposed algorithm could determine whether any given Plaintiff had exceeded the Lifetime

Cumulative Exposure to NDMA and/or NDEA that Plaintiffs have proposed to define class membership.

16. Levels of NDMA and/or NDEA in valsartan API varied as a function of manufacturer, calendar date, and specific lot. Plaintiffs' proposal for an Lifetime Cumulative Exposure threshold to determine class membership ignores this essential quantitative information regarding NDMA and NDEA content, which they claim is ascertainable, in favor of a standard that excludes information on actual NDMA and/or NDEA content (*i.e.*, months of exposure to a particular manufacturer's valsartan API irrespective of manufactured lot). Plaintiffs' approach effectively – and incorrectly – assumes that the amount of NDMA and/or NDEA in any manufacturer's API was (a) above the FDA ADI threshold, and (b) constant over time and exhibiting no lot-to-lot variability. Thus, Plaintiffs' claim that "*determination of whether the class member consumed a Lifetime Cumulative Threshold sufficient for class membership is based on objective and ascertainable factors"* is false. Third Amended Medical Monitoring Class Action Complaint ¶¶ 540-541.

17. Contrary to Plaintiffs' claim that they will specify a Lifetime Cumulative Exposure to NDMA and/or NDEA as a "threshold" for medical monitoring (*i.e.*, a requirement for medical monitoring would be triggered by ingestion of a certain number of micrograms of NDMA or NDEA in valsartan containing products), Plaintiffs instead substitute a criterion for ingestion of a certain number of months of valsartan products of known doses and formulations from particular manufacturers' APIs. Consequently, despite Plaintiffs' claim that NDMA and/or NDEA exposures are ascertainable, Plaintiffs' proposal for a Lifetime Cumulative Exposure threshold discards Plaintiff-specific information regarding NDMA and NDEA exposure (*i.e.*, specific lots of finished medication containing a specific lot of a specific manufacturer's API at specific points in time) in favor of a standard that would inevitably introduce gross errors into exposure estimates for individual Plaintiffs.

18. For these reasons, and others, Plaintiffs' formulaic claim that "*Plaintiff X consumed a Cumulative Lifetime Threshold of at least XXX units"* [of NDMA, NDEA, or other nitrosamine] and, as a result, "*suffered cellular and genetic injury that creates and/or increases the risk that Plaintiff will develop cancer*" cannot possibly be met by applying the algorithm that Plaintiffs propose. Third Amended Medical Monitoring Class Action Complaint ¶¶ 12-28.

Plaintiffs' claimed dose/duration/API thresholds for medical monitoring for cancer lack a rational medical or scientific basis

19. As above, Plaintiffs fail to articulate a basis for the particular dose/duration/API thresholds proposed for medical monitoring, as no medical or scientific basis for these thresholds is stated either in the Third Amended Medical Monitoring Class Action Complaint or in the Memorandum of Law in Support of Plaintiffs' Motion for Class Certification. In particular, no reliable method is provided by which cumulative exposures to NDMA and/or NDEA attributable to ingestion of valsartan products could be ascertained from the proposed dose/duration/API thresholds. In the absence of a reliable method to "translate" the number of months of ingestion of a particular valsartan dose from a particular manufacturer's API to a cumulative exposure to a certain number of micrograms of NDMA

and/or NDEA, it is impossible to use the body of dietary epidemiology studies referenced by Dr. Madigan to infer a change in cancer risk among Plaintiffs who ingested valsartan products, since the studies referenced by Dr. Madigan each relate cancer risk to the number of micrograms of lifetime cumulative exposure to NDMA and/or NDEA.

20. For example, Plaintiffs' definition of Lifetime Cumulative Exposure proposes that a person who ingested products containing valsartan at a dose of 320 mg, formulated from ZHP API for three months, Hetero API for 18 months, or Mylan and/or Aurobindo API for 54 months, would qualify as a class member. Third Amended Medical Monitoring Class Action Complaint ¶¶ 540-541. First and foremost, the fact that the NDMA and NDEA content of different valsartan products varied widely across different manufactured lots of valsartan API, even from the same manufacturer, and that many lots of valsartan API contained levels of NDMA and/or NDEA that were either below the limits of detection or below the FDA threshold of Acceptable Daily Intake (ADI), obviates the reliability of this approach.

21. The above fatal logical flaw notwithstanding, it can be calculated that ingestion of ZHP API from the manufactured lot containing the highest level of NDMA measured by the FDA in a 320 mg valsartan product (Prinston Pharmaceutical, 20.19 ug/tablet) for 3 months would correspond to 1,841 ug of NDMA (3 months x 30.4 days/month x 20.19 ug NDMA/tablet).[1] That is, the maximum hypothetical exposure to NDMA as a consequence of ingesting 3 months of ZHP valsartan API is 1,841 ug. This amount is roughly similar to, although lower than, the lowest Lifetime Cumulative Exposure to NDMA that Dr. Madigan has stated is associated with an increased risk for *any* type of cancer in human beings (1,962 ug NDMA for gastric cancer). Report of Dr. David Madigan July 7, 2021 ¶¶ 23, 33 and Table 1. Similarly, it can calculated that ingestion of Hetero API from the manufactured lot containing the highest level of NDMA measured by the FDA in a 320 mg valsartan product (Hetero Labs, 0.44 ug/tablet) for 18 months would correspond to 241 ug of NDMA (18 months x 30.4 days/month x 0.44 ug NDMA/tablet).[1] That is, the maximum hypothetical exposure to NDMA as a consequence of ingesting 18 months of Hetero valsartan API is 241 ug. This amount is 8.2-times *lower* than the lowest Lifetime Cumulative Exposure to NDMA that Dr. Madigan has stated is associated with an increased risk for *any* type of cancer in human beings (1,962 ug NDMA for gastric cancer). Report of Dr. David Madigan July 7, 2021 ¶¶ 23, 33 and Table 1. Given Plaintiffs' failure to articulate a reliable medical or scientific basis for their claimed dose/duration/API thresholds, or to demonstrate how such thresholds correspond to actual NDMA exposures (which they do not), for the purposes of this report I have assumed that Plaintiffs' claim is that any individual exposed to at least 1,962 ug of NDMA as a consequence of ingesting valsartan products is at an increased risk for developing cancer and therefore requires medical monitoring.

22. In an analogous manner, given that valsartan products manufactured by Mylan and Aurobindo did not contain detectable levels of NDMA, it can be calculated that ingestion of the API that would yield the highest cumulative amount of NDEA through application of Plaintiffs' algorithm would correspond to the NDEA level measured by the FDA in a 320 mg valsartan product (Mylan, 0.38 ug/tablet) for 54 months. This would correspond to 624 ug of NDEA (54 months x 30.4 days/month x 0.38 ug NDEA/tablet).[1] This level of exposure is 4-

times *lower* than the lowest Lifetime Cumulative Exposure to NDEA that Dr. Madigan has claimed is associated with an increased risk for any type of cancer in human beings (2,520 ug NDEA for pancreas cancer).

23. It can also be calculated that ingestion of ZHP API from the manufactured lot containing the highest level of NDEA measured by the FDA in a 160 mg valsartan product (Torrent Pharmaceuticals, 1.31 ug/tablet) for 6 months (per Plaintiffs proposed threshold algorithm) would correspond to 239 ug of NDEA (6 months x 30.4 days/month x 1.31 ug NDEA/tablet).[1] This level of exposure is 10.5-times *lower* than the lowest Lifetime Cumulative Exposure to NDEA that Dr. Madigan has claimed is associated with an increased risk for any type of cancer in human beings. Report of Dr. Madigan July 7, 2021 Table 1.

24. Thus, through their proposed approach to define class membership, Plaintiffs appear to suggest that exposure to NDEA at levels 4-fold to 10.5-fold *lower* the lowest Lifetime Cumulative Exposure to NDEA associated with a statistically significant increased risk of any type of cancer in human beings nevertheless requires lifetime medical monitoring for the development of cancer. Given that application of Plaintiffs' proposed Lifetime Cumulative Exposure criterion for NDEA is unsupported by medical or scientific evidence and fails to meet any reasonable test of logic, to the extent that Plaintiffs' claim that ingestion of valsartan products containing nitrosamines warrants lifetime medical monitoring is based on medical evidence, I conclude that their claim is based upon estimated exposures to NDMA, not NDEA.

25. Indeed, given that Mylan and Aurobindo valsartan APIs did not contain detectable levels of NDMA, and that the maximum theoretical exposures to NDEA from Mylan and Aurobindo APIs were several times *lower* than the lowest Lifetime Cumulative Exposure to NDEA that Dr. Madigan has claimed is associated with an increased risk for *any* type of cancer in human beings, it is entirely unclear to me how exposure to these manufacturer's APIs could logically be considered to warrant lifetime medical monitoring, as proposed by Plaintiffs' Lifetime Cumulative Threshold algorithm.

26. The maximum period of time that valsartan products potentially containing NDMA were available on the U.S. market was 51 months (9/29/2014-12/31/2018). The maximum period of time that valsartan products potentially containing NDEA were available on the U.S. market was 75 months (9/21/2012-12/31/2018). Report of Dr. Chodosh August 2, 2021 ¶¶ 137, 140. In light of these facts, which to my knowledge are uncontested, I find it incomprehensible that the durations of exposure to valsartan products potentially containing NDMA or NDEA that are proposed by Plaintiffs' algorithm to trigger lifetime medical monitoring include durations as long as 108, 128, 216 and 432 months. The inclusion of such proposed durations of exposure lacks even a modicum of common sense and appears to be predicated upon the existence of a universe in which even the most elementary facts need not be considered. This alone highlights the chasm separating Plaintiffs' proposed algorithm for determining Lifetime Cumulative Exposure thresholds and any reasonable – or even vaguely logical – understanding of the facts regarding potential

exposures to NDMA and/or NDEA through the ingestion of valsartan products that were even physically possible.

Plaintiffs' claimed dose/duration/API thresholds for medical monitoring for cancer are orders of magnitude lower than levels of NDMA, NDEA and other nitrosamines to which human beings are routinely exposed

27. Human exposures to *N*-nitrosamines can be classified as either exogenous or endogenous. Exogenous exposures involve pre-formed NDMA, whereas endogenous exposures entail the synthesis of *N*-nitrosamines, including NDMA, within the body. Significant sources of exogenous exposure to NDMA include food, indoor air (*i.e.*, environmental tobacco smoke), drinking water, beverage alcohol, tobacco, consumer products, and occupational exposures in certain industries. Report of Dr. Chodosh August 2, 2021 ¶ 82.

28. I agree with Dr. Madigan's assessment that "*the U.S. Food and Drug Administration has indicated that levels of NDMA up to 0.096 ug/day and levels of NDEA up to 0.0265 ug/day are safe*".[1,2] Report of Dr. David Madigan July 7, 2021 ¶ 6. Of note, however, the FDA Acceptable Daily Intake (ADI) for NDMA of 0.096 ug/d corresponds to 35 ug per year and 2,454 ug over a 70-year lifetime. Consequently, it is difficult to reconcile the FDA's assessment that lifetime cumulative exposure to 2,454 ug of NDMA is safe (an assessment with which Plaintiffs' expert Dr. Madigan agrees) with Plaintiffs' claim that lifetime cumulative exposure to 1,962 ug of NDMA significantly raises the risk of cancer and warrants lifetime medical monitoring for cancer. Moreover, given that ingestion of 320 mg valsartan products containing ZHP API for 3 months corresponds to a maximum amount of NDMA that is even lower (1,841 ug), I cannot discern a rational scientific or medical basis for Plaintiffs' claim that exposure to this level of NDMA should trigger a requirement for lifetime medical monitoring for cancer. Even more bizarre is Plaintiffs' claim, based upon ingestion of 320 mg valsartan products containing Hetero API for 18 months, that exposure to as little as 241 ug of NDMA should trigger lifetime medical monitoring for cancer, given that this level is more than 10-times *lower* than the lifetime cumulative exposure to NDMA that the FDA considers to be safe.

29. In an analogous manner, the FDA Acceptable Daily Intake (ADI) for NDEA of 0.0265 ug/d corresponds to 678 ug over a 70-year lifetime. Consequently, it is difficult to reconcile the FDA's assessment that lifetime cumulative exposure to 678 ug of NDEA is safe (an assessment with which Plaintiffs' expert Dr. Madigan agrees) with Plaintiffs' claim that lifetime cumulative exposure to as little as 624 ug of NDEA, or even 239 ug (or lower), requires lifetime medical monitoring for cancer.

30. As detailed in my report, estimates of dietary intake of NDMA (excluding beer or tobacco) from seven studies[3-9] yield an average cumulative exposure of 4,190 ug over a 70-year lifetime for a 70 kg person. Liteplo et al.[10] estimated ranges for intake of NDMA from food (excluding tobacco or beer/whiskey) for persons aged 20-59 years at 7,700 – 19,700 ug over a 70-year lifetime for a 70 kg person. This indicates that daily dietary intake of NDMA, even excluding tobacco and beer, may range from 1.7 – 8.0-times higher than the FDA ADI[10], and

2.1 – 10.0-times higher than the 1,962 ug threshold apparently proposed by Plaintiffs for medical monitoring. Report of Dr. Chodosh August 2, 2021 ¶¶ 87-88.

31. In light of estimates of lifetime cumulative exposure to NDMA attributable to dietary intake ranging from 4,190 – 19,700 ug, it is difficult to discern a rational basis for Plaintiffs' claim that lifetime cumulative exposure to as little as 1,962 ug of NDMA should require medical monitoring given that most, if not all, persons in the United States are likely exposed to substantially higher levels of NDMA simply through the foods that they eat.

32. Liteplo *et al.* reported "*reasonable worst-case estimates*" for daily intake of NDMA from food, water, and outdoor air (excluding tobacco or beer/whiskey) for ages 20-59 years ranging from 0.005 – 0.016 ug/kg/d.[10] This would correspond to 8,950 – 28,600 ug of exposure to NDMA over a 70-year lifetime for a 70 kg person. These estimates of exposure to NDMA through food, water and air are 3.6 – 11.7-times higher than the FDA ADI. Exposures would be higher for persons drinking beer and/or whiskey, and might be up to an order of magnitude higher for those with tobacco exposures.[10] Report of Dr. Chodosh August 2, 2021 ¶ 91.

33. As above, in light of estimates of lifetime cumulative exposure to NDMA attributable to food, air and water ranging from 8,950 – 28,600 ug, Plaintiffs' claim that lifetime cumulative exposure to as little as 1,962 ug of NDMA should require medical monitoring lacks a reasonable scientific basis, given that most, if not all, persons in the United States are likely exposed to substantially higher levels of NDMA simply by eating, drinking and breathing.

34. Beyond exogenous exposures to NDMA from food, water, air, tobacco, beer, whiskey, and consumer products, endogenous exposures to NDMA are increasingly appreciated to be a major source – if not the major source – of exposure to NDMA and other nitrosamines.[3,11-14] Indeed, NDMA and other nitrosamines are formed endogenously within the body as a consequence of normal human physiology.[3,11-16] NDMA and other nitrosamines are formed endogenously from precursor compounds contained in food, are formed endogenously in the gastrointestinal (GI) tract due to the metabolism of red meat in the presence of bacteria in the GI tract, and may also be formed by acid-catalyzed nitrosation in the stomach and by enzyme-catalyzed nitrosation[10,13,17-27] Report of Dr. Chodosh August 2, 2021 ¶¶ 95-98.

35. Average daily exposure to endogenously produced NDMA is likely on the order of 15 ug/kg/d, which would correspond to 1,050 ug/d for a 70 kg person, and 26,800,000 ug of NDMA over a 70-year lifetime.[3] This level of exposure to endogenous NDMA is approximately 1,875-times higher than the highest estimate of daily exposure to exogenous NDMA in food, drinking water and air[10], and nearly 11,000-times higher than the FDA ADI of 0.096 ug/d. Thus, endogenous exposures to NDMA are likely to be three orders of magnitude higher than exposures to preformed NDMA in food, air and water combined, and four orders of magnitude higher than a lifetime cumulative exposure to NDMA that the FDA considers to be "*safe*".[1,2] These data, and others, strongly suggest that the greatest human exposure to NDMA, by far, occurs as a consequence of endogenous formation of NDMA, not dietary intake, and these levels are several orders of magnitude higher than the Lifetime Cumulative Exposure threshold proposed by Plaintiffs to trigger a requirement for

medical monitoring for cancer. Put simply, Plaintiffs' proposal to use a Lifetime Cumulative Exposure to NDMA as a trigger for medical monitoring for cancer that is 10,000-times lower than that to which human beings are routinely exposed as a consequence of normal physiology is illogical and contradicted by medical and scientific evidence. Report of Dr. Chodosh August 2, 2021 ¶¶ 95-98.

36. As discussed in detail in my report of August 2, 2021, the *maximum* theoretical total exposure to NDMA to which a Plaintiff could conceivably have been exposed due to ingestion of any combination of valsartan products was 26,635 ug. I arrived at this estimate using NDMA testing data from the FDA for valsartan products,[1] coupled with date ranges that different valsartan products were approved for sale in the U.S.[28]. Specifically, I identified the valsartan product with the highest level of NDMA that was available on the U.S. market for each day from 9/29/2014-12/31/2018. This permitted me to calculate the theoretical maximum exposure that conceivably could have occurred if, on any given day, a theoretical Plaintiff took the valsartan product available on the market on that day that had the highest FDA-measured level of NDMA, irrespective of manufacturer, from the first date that a valsartan product potentially containing NDMA was first approved for sale on the U.S. market, until the last recall date of 12/31/2018. My conservative estimate additionally included the assumptions that: (a) the Plaintiff filled a prescription on the first day that a valsartan product potentially containing NDMA was approved for sale in the U.S. under the process change; (b) the Plaintiff continued to take valsartan products for the entire period from 9/29/2014-12/31/2018; (c) each and every valsartan tablet that they received and ingested over the entire 4.4 year interval contained the highest FDA-measured dose of NDMA in any valsartan product available on the U.S. market on that day; and (d) on the day of product recall they filled a 90-day prescription for the valsartan product containing the highest FDA-measured dose of NDMA available on the U.S. market on that day, and continued to take that product for the entire 90-day duration of the prescription. If all of these assumptions were met, the maximum theoretical cumulative NDMA exposure due to ingestion of valsartan products would have been 26,635 ug. Using an analogous approach, it can be estimated that the maximum theoretical cumulative exposure to NDEA to which a Plaintiff could conceivably have been exposed due to ingestion of valsartan products was 2,157 ug. Report of Dr. Chodosh August 2, 2021 ¶¶ 132-137, 142, 148-153.

37. Given the above estimates of cumulative lifetime exposure to NDMA due to endogenous production of 26,800,000 ug over a 70-year lifetime, it is evident that the highest conceivable exposure to NDMA by ingestion of valsartan products is still 1,000-times *lower* than exposure due to endogenous NDMA production. That is, the maximum theoretical cumulative exposure to NDMA from valsartan products represents – at most – just 0.1% of estimated total lifetime exposure to endogenously produced NDMA. Report of Dr. Chodosh August 2, 2021 ¶¶ 132-137, 142, 148-153. Similarly, the Lifetime Cumulative Exposure threshold for NDMA that Plaintiffs propose as a threshold for medical monitoring is – at most – just 0.01% of estimated total lifetime exposure to endogenously produced NDMA.

38. In light of the above, Plaintiffs' claim that exposure to as little as 1,962 ug (or lower) of NDMA through ingestion of valsartan-containing products is sufficient to warrant lifetime

medical monitoring for the development of cancer is notable for the fact that this level of exposure is: (a) lower than the lifetime cumulative exposure to NDMA that FDA considers to be safe; (b) 2.1 – 10.0-times lower than dietary exposures to preformed NDMA; (c) 4.6 – 14.6-times lower than exposures to NDMA in food, air and water; and (d) >13,000-times lower than estimates of cumulative lifetime exposure to NDMA due to endogenous production. That is, the level of NDMA exposure through ingestion of valsartan products claimed by Plaintiffs to be sufficient to warrant lifetime medical monitoring is more than four orders of magnitude lower than the levels of NDMA to which all human beings are routinely exposed as a consequence of normal physiology. If all human beings are exposed to levels of NDMA that are many thousands of times higher than the threshold for medical monitoring proposed by Plaintiffs, such a threshold fails the test of common sense.

Even the maximum hypothetical exposures to NDMA and NDEA as a consequence of ingesting valsartan containing products do not cause cancer

39. Although NDMA and NDEA are known carcinogens in laboratory animals, they are not known carcinogens in humans. No type of cancer in humans has been conclusively demonstrated to result from exposure to NDMA or NDEA. Furthermore, there are no dose-response data from which to infer levels of exposure to NDMA or NDEA that might cause cancers in human beings. Consistent with this, no regulatory agency has classified NDMA or NDEA as a known human carcinogen. As such, there is no reliable scientific basis to conclude that either NDMA or NDEA is a human carcinogen. Report of Dr. Chodosh August 2, 2021 ¶ 115.

40. As described in detail in my report of August 2, 2021, the maximum hypothetical exposures to NDMA and NDEA that conceivably could have occurred in Plaintiffs as a consequence of ingesting valsartan products do not cause cancer in human beings. Specifically, even these maximum hypothetical exposures are thousands of times *lower* than the lowest doses of NDMA or NDEA reported to cause cancer in laboratory animals. Report of Dr. Chodosh August 2, 2021 ¶¶ 132-137, 142, 154-156.

41. As above, the maximum theoretical total exposure to NDMA to which a Plaintiff could conceivably have been exposed due to ingestion of any combination of valsartan products was 26,635 ug. This maximum exposure is approximately 2,280-times *lower* than the lowest dose of NDMA shown to cause a detectable increase in cancer in rats, 0.034 mg/kg/d, when administered over a lifetime. This dosage in rats would correspond to an exposure of 60,850,650 ug over a 70-year lifetime for a 70 kg person. Moreover, Plaintiffs' apparent claim that exposure to as little as 1,962 ug of NDMA through ingestion of valsartan products is sufficient to warrant lifetime medical monitoring for cancer is remarkable insofar as this level of NDMA exposure is 31,000-times lower than the lowest dose of NDMA shown to cause a detectable increase in cancer in the most NDMA-sensitive tissue in rats. In an analogous manner, Plaintiffs' claim (for Hetero API) that exposure to as little as 241 ug of NDMA through ingestion of valsartan products is sufficient to warrant lifetime medical monitoring for cancer borders on incomprehensible, given that this level of NDMA exposure is more than 250,000-times *lower* than the lowest dose of NDMA shown to cause a

detectable increase in cancer in the most NDMA-sensitive tissue in rats. Report of Dr. Chodosh August 2, 2021 ¶¶ 132-137, 142, 154-156.

42. As above, the maximum theoretical cumulative exposure to NDEA to which a Plaintiff could conceivably have been exposed due to ingestion of any combination of valsartan products was 2,157 ug. By way of comparison, rats treated with doses of NDEA as high as 0.0108 mg/kg/d over the course of their lifetime show no detectable increase in cancer. This no observed effect level for NDEA in rats corresponds to an NDEA exposure of 19,329,030 ug over a 70-year lifetime for a 70 kg person. Thus, the 2,157 ug maximum theoretical total exposure to NDEA to which a Plaintiff could conceivably have been exposed due to ingestion of valsartan products is nearly 9,000-times *lower* than a dose of NDEA that does not cause a detectable increase in cancer in rats when administered over a lifetime. Thus, Plaintiffs' apparent claim that exposure to as little as 624 ug of NDEA through ingestion of valsartan containing products is sufficient to warrant lifetime medical monitoring for cancer is remarkable insofar as this level of NDEA exposure is 31,000-fold *lower* than a dose of NDEA that does not cause any detectable increase in cancer in rats when administered over a lifetime. ¶¶ 140-142, 158-160 Report of Dr. Chodosh August 2, 2021.

43. For the reasons discussed in detail in my report of August 2, 2021, and briefly summarized above, it is my conclusion, to a reasonable degree of medical and scientific certainty, that exposure to NDMA and/or NDEA in valsartan, at the doses to which Plaintiffs were potentially exposed, and for the durations to which Plaintiffs were potentially exposed, would not cause cancer in human beings. Given that conclusion, it would be illogical to posit that levels of exposure to NDMA and NDEA that are substantially lower are sufficient to warrant lifetime medical monitoring for the development of cancer, as Plaintiffs have proposed.

The basis for Plaintiffs' proposed Lifetime Cumulative Exposure threshold for medical monitoring for cancer is subjective, arbitrary, unscientific and unreliable

44. Plaintiffs claim that reports from Drs. Madigan and Panigrahy "*demonstrate, using dietary and other studies, that there is a threshold NDMA Lifetime Cumulative Exposure associated with statistically significant increased risks of developing cancers.*" Plaintiffs then propose that exceeding this Lifetime Cumulative Exposure threshold for NDMA and NDEA exposure as a consequence of ingesting valsartan products should trigger a requirement for lifetime medical monitoring for the development of cancer. Memorandum of Law in Support of Plaintiffs' Motion for Class Certification §§ II.B.2, V.A.4.

45. Specifically, Plaintiffs claim elevated risks for the development of esophageal, stomach, colorectal/intestinal, liver, lung, bladder, blood, pancreatic, and prostate cancers for those Plaintiffs who consumed valsartan products in amounts greater than the Lifetime Cumulative Threshold that they propose. Evaluation of the reports of Drs. Panigrahy and Madigan reveals that these Lifetime Cumulative Exposure thresholds for NDMA and NDEA are primarily based upon dietary epidemiology studies, with supplementation from a single occupational study of inhalation exposures in rubber factory workers for those cancers that were not found to have significantly elevated risks associated with NDMA or NDEA in

dietary studies. However, as detailed below, Plaintiffs' approach is subjective, arbitrary, unscientific and unreliable, if not fatally flawed.

46. First, it bears noting that the only existing epidemiological data to examine human exposure to valsartan containing NDMA and/or NDEA support the conclusion that exposure to NDMA and/or NDEA at the doses to which Plaintiffs were potentially exposed, and for the durations to which Plaintiffs were potentially exposed, would not cause cancer in human beings. In particular, Gomm et al.[29] and Pottegard et al.[30] showed no increase in the risk of bladder cancer, breast cancer, colorectal cancer, kidney cancer, lung cancer, melanoma, prostate cancer, or uterine cancer. Gomm found a statistically significant association between exposure to valsartan containing the impurity and liver cancer, but no dose-dependent effect was observed, the study did not control for other risk factors for liver cancer, and it did not correct for multiple testing (*i.e.*, testing for an association for multiple types of cancer, one type at a time), which most likely would have rendered the result statistically insignificant. Most importantly, the authors themselves concluded from their study that: "*Causality cannot be inferred.*" Report of Dr. Chodosh August 2, 2021 ¶¶ 171-173 and at 63.

47. Second, for the many reasons discussed at length in my report, dietary epidemiology studies are an unreliable basis for identifying an amount of NDMA or NDEA associated with an increased risk of cancer in human beings.[31] Both dietary epidemiology studies and occupational inhalation studies are subject to a variety of important limitations that preclude reliable interpretations regarding the potential carcinogenic effects of ingested NDMA and NDEA in human populations. Report of Dr. Chodosh August 2, 2021 ¶¶ 113-121.

48. For example, dietary epidemiology studies typically rely upon observational study designs based on self-reported dietary behavior. Beyond the fact that observational studies can never fully control for unrecognized bias and confounding, perhaps the major drawback to observational dietary studies is measurement error in dietary assessment.[31] Most importantly, the actual NDMA content in foods to which study subjects are exposed is rarely, if ever, actually measured in observational dietary studies. Rather, NDMA intake is inferred based on prior measurements of NDMA content in different foods, coupled with assessments of dietary intake that are typically based on self-reporting. The use of pre-existing data on NDMA content in food is itself associated with a variety of limitations and potential errors, including variation in NDMA levels even in the "same" food from different geographical regions, or grown, prepared or preserved in different ways.[31] Furthermore, dietary assessments based on self-reporting are notoriously inaccurate (*e.g.*, do not correspond to actual intake), irrespective of whether the dietary assessment tool itself is "validated", insofar as the validation of such tools generally refers to the presence of a correlation between dietary intake estimates based on the use of different assessment tools.[31] Indeed, the complexity of human dietary patterns and practices is difficult to capture within a food questionnaire, self-reporting of foods consumed as well as portion sizes is subject to marked distortion and bias, and food histories queried many years after the meaningful exposures likely occurred (*i.e.*, with regard to cancer development) are intrinsically limited. For these reasons, and others, the actual dietary NDMA exposures of

participants in observational dietary epidemiology studies is almost always unknown. Report of Dr. Chodosh August 2, 2021 ¶ 117.

49. An additional critical drawback of dietary epidemiology studies is related to the complexity of foodstuffs. Foods typically contain thousands of chemical compounds, only a small minority of which may be measured. Even with respect to a single family of compounds, such as nitrosamines, multiple members of a family of molecules may be present in differing amounts in different foods. Consequently, there is a fundamental statistical limitation of interpreting correlations between the estimated dietary intake of a particular chemical (*e.g.*, NDMA) and an outcome, because there are simply far more variables with respect to food composition than there are observations of outcomes. That is, there are not enough study participants, and not enough measures of the many different chemical compounds in the food they consumed (if any), to reliably determine the effect of a single chemical compound and disentangle these hypothetical effects from the many thousands of other chemical compounds present in varying amounts. For example, an observed association of NDMA with a particular clinical outcome could result if estimated NDMA intake was merely a marker for (*i.e.*, correlated with) a different, unknown or unmeasured exposure – even another nitrosamine. If the estimated NDMA dietary intake for a particular individual is high because they eat large amounts of bacon, there are obviously many other chemical compounds in bacon besides NDMA, and there are likely many things that differ between people who eat large amounts of bacon and those who do not, besides NDMA intake. Still more complex, if a person has a high estimated NDMA intake due to consumption of processed or grilled red meat, effects due to consumption of preformed NDMA cannot reasonably be separated from effects due to the endogenous generation of NDMA in the GI tract due to the presence of heme in red meat, or to the generation of preformed carcinogens such as heterocyclic amines at high temperatures. These factors, and others, impair the reliability with which inferences can be drawn between the dietary consumption of a single constituent in foods and a particular clinical outcome, and these limitations are compounded further when the actual constituents consumed are not accurately measured, if at all. Report of Dr. Chodosh August 2, 2021 ¶ 118.

50. In light of the above considerations it is evident that available dietary epidemiological studies focused on NDMA (a) do not actually measure NDMA intake in study participants; (b) do not measure the amounts of the thousands of other chemical constituents present in the food consumed by study participants; (c) cannot reliably distinguish potential effects of NDMA from the many other constituents in food; (d) are unlikely to accurately measure food intake of study participants, irrespective of the chemical constituents contained within it; (e) do not measure food intake during the periods of time in which cancers actually form; (f) cannot rule out potential confounders and biases as explanations for any observed associations; and (g) cannot demonstrate cause and effect. For these reasons, and others, there is no medical or scientific basis by which existing dietary epidemiological studies of NDMA could reliably attribute any observed differences in cancers among study participants to NDMA. Report of Dr. Chodosh August 2, 2021 ¶ 119.

51. As outlined below, the many limitations of dietary epidemiology studies of the potential effects of NDMA and/or NDEA on cancer risk are starkly illustrated by the conclusions reached by Plaintiffs from the reports of Drs. Panigrahy and Madigan. Further, these inherent limitations are accentuated by subjective and arbitrary choices that Plaintiffs make in selecting certain pieces of data to highlight while excluding the much larger body of data that undercuts, if not invalidates, their conclusions.

52. In his report, Dr. Madigan relies upon dietary epidemiology studies to derive Lifetime Cumulative Exposures to NDMA that he considers to be associated with statistically significant increased risks for each of several particular cancers. Specifically, in Table 1 of his report Dr. Madigan's lists 25 studies for seven different cancer types from which he calculated Lifetime Cumulative Exposure levels for NDMA. This analysis, in turn, served as the basis for Plaintiffs' proposed threshold for medical monitoring. Report of Dr. David Madigan July 7, 2021 ¶¶ 23-26, 33, Table 1.

53. First, and perhaps most tellingly, of the 25 studies listed by Dr. Madigan, fewer than half (12) are statistically significant. Indeed, for three of the seven cancers evaluated by Dr. Madigan (bladder cancer, prostate cancer and pancreas cancer), no statistically significant increased risk associated with NDMA dietary lifetime cumulative exposure was shown in *any* study. In addition, for gastric cancer and esophageal cancer, the majority of studies listedby Dr. Madigan did not show a statistically significant increased risk of cancer associated with dietary lifetime cumulative exposure to NDMA (gastric cancer: five of nine studies not statistically significant; esophageal cancer: three of four studies not statistically significant). Strikingly, dietary lifetime cumulative exposure to NDMA is not associated with a statistically significant increased risk for cancer of the liver, even though this is the organ widely accepted as the most sensitive to the carcinogenic effects of NDMA in animals. In my opinion, the most reasonable scientific interpretation of these data would be that there is not a reproducible association between dietary lifetime cumulative exposure to NDMA and cancers of the stomach, esophagus, bladder, prostate, pancreas or liver. Report of Dr. David Madigan July 7, 2021 Table 1.

54. Even when considering the only two cancers (of the seven cancers that Dr. Madigan evaluated in Table 1), for which at least half of the studies showed a statistically significant association with dietary lifetime cumulative exposure to NDMA, these values ranged from 6,114 – 16,363 ug for lung cancer (average 11,238 ug) and 3,343 – 27,628 ug for colon/colorectal cancer (average 15,486 ug). These average levels are 5.7 – 7.9-times higher than the threshold for NDMA exposure selected by Plaintiffs for lifetime medical monitoring. Indeed, even the average of all of the statistically significant lifetime cumulative exposure levels listed by Dr. Madigan in Table 1 is greater than 8,000 ug of NDMA, which is more than 4-times higher than the threshold proposed by Plaintiffs. Report of Dr. David Madigan July 7, 2021 Table 1.

55. Dr. Madigan notes lifetime cumulative exposures to NDMA associated with increased risks of cancer "*as low as*" 1,962 ug (Larsson, gastric cancer), 3,343 ug (Loh, rectal cancer), 4,235 ug (Keszei, esophageal cancer) and 4,303 ug (DeStefani, lung cancer), as well as lifetime

cumulative exposure to NDEA associated with increased risks of cancer as low as 2,520 ug for pancreas cancer (Zheng). Report of Dr. David Madigan July 7, 2021 ¶¶ 23-26, 33, Table 1. However, as outlined above, highlighting the handful of findings that are the least incongruous with Plaintiffs' selected monitoring threshold, while ignoring the much larger body of data that flatly contradicts it, lacks objectivity and is unscientific. Report of Dr. David Madigan July 7, 2021 ¶¶ 23-26, 33, Table 1.

56. In his report, Dr. Panigrahy refers to lifetime cumulative dietary exposures to NDMA that were associated with statistically significant increased risks of certain cancers in some published dietary epidemiology studies: gastric cancer (DeStefani 5,913 ug; La Vecchia 4,161 ug), colorectal cancer (Knekt 1,160 ug; Larsson 4,161 ug; Loh 2,759 ug) and lung cancer (DeStefani 5,913 ug; Goodman 15,330 ug). Report of Dr. Dipak Panigrahy July 6, 2021 at 90.

57. Given Dr. Panigrahy's reasoning that these studies identify dietary lifetime cumulative exposures to NDMA that are associated with statistically significant increased risks of certain cancers, the selection by Plaintiffs of a Lifetime Cumulative Exposure threshold for medical monitoring that corresponds to 1,962 ug NDMA is arbitrary and subjective. For example, this threshold is several times lower than six of the seven dietary lifetime cumulative exposure levels that Dr. Panigrahy cites as being associated with an increased risk of cancer. Indeed, the average of the seven NDMA dietary lifetime cumulative exposure levels that Dr. Panigrahy cites is 5,628 ug NDMA – a value that is nearly 3-fold higher than the 1,962 ug threshold selected by Plaintiffs.

58. It is also remarkable that Drs. Panigrahy and Madigan identify different lifetime cumulative exposure thresholds for dietary NDMA exposures associated with statistically significant increases in cancer risk for the very same cancers (*i.e.*, gastric: 5,913 ug and 4,161 ug from Panigrahy vs. 1,962 from Madigan; colorectal: 1,160 ug, 4,161 ug and 2,759 ug from Panigrahy vs. 3,343 from Madigan; lung: 5,913 ug and 15,330 ug from Panigrahy vs. 4,303 ug from Madigan). Second, it is notable that Plaintiffs' experts each choose different studies from which to estimate lifetime cumulative dietary NDMA exposure levels associated with an increased risk of being diagnosed with the same cancer. Third, and most remarkably, Drs. Panigrahy and Madigan "identify" different lifetime cumulative exposure thresholds for dietary NDMA exposures associated with statistically significant increases in cancer risk for the same cancers – even for those estimates that appear to be based upon the same study (*i.e.*, DeStefani lung cancer: 5,913 ug from Panigrahy vs. 4,303 ug from Madigan; Loh colorectal cancer: 2,759 ug from Panigrahy vs. 3,343 ug from Madigan). That these two Plaintiffs' experts, upon whom Plaintiffs rely for establishing a Lifetime Cumulative Exposure threshold for medical monitoring, arrive at different exposure thresholds for the same cancers, and in some cases from the same studies, indicates to me that the methodology employed by Plaintiffs' experts is subjective and unreliable.

59. As above, Plaintiffs claim elevated risks for developing esophageal, stomach, colorectal, liver, lung, bladder, blood, pancreatic, and prostate cancers for those Plaintiffs who consumed valsartan products in amounts greater than the Lifetime Cumulative Exposure threshold that they propose. However, the dietary epidemiology studies cited by Drs.

Madigan and Panigrahy in support of this fail to show statistically significant associations with dietary NDMA exposure for bladder cancer, prostate cancer, pancreas cancer, liver cancer, or blood cancers – that is, for five of the nine cancers claimed. For two additional cancers, a majority of studies failed to show a statistically significant association with dietary lifetime cumulative exposure to NDMA.

60. Consequently, to support Plaintiffs' claims regarding NDMA and cancer risk, Drs. Madigan and Panigrahy turn to an occupational inhalation study of workers in the rubber manufacturing industry. Specifically, Dr. Madigan concludes based upon the occupational exposure study of Hidajat et al. that "*cumulative exposure to greater than 7,514 ug of NDMA*[27] *statistically significantly increases one's risk of developing the following cancers – bladder, lung, stomach, multiple myeloma, esophageal, prostate, and prostate [sic]. Cumulative exposure greater than 14,319 ug of NDMA adds leukemia, lymphoma, and liver to the list.*" Report of Dr. David Madigan July 7, 2021 ¶ 34.

61. As noted in my report of August 2, 2021, occupational studies related to the effects of *inhaled* NDMA exposure are subject to important limitations that preclude reliable inferences regarding the effects of low-dose *oral* consumption of NDMA. These include the different route of exposure (inhalation vs. oral ingestion), higher levels of exposure, simultaneous exposure to multiple different potentially carcinogenic substances (*e.g.*, rubber dust, rubber fumes, NDMA, and other nitrosamines in rubber and tire manufacturing workers), and lack of information on important confounders (*e.g.*, smoking). Report of Dr. Chodosh August 2, 2021 ¶ 121. Thus, for the seven of nine claimed cancer associations with NDMA that lack reliable support from dietary epidemiology studies, Drs. Panigrahy and Madigan's reliance on an occupational inhalation exposure study is scientifically unfounded and unreliable. Moreover, the lifetime cumulative exposure thresholds identified by Dr. Madigan using this occupational study (7,514 ug and 14,319 ug) are 3.8 – 7.2-times higher than the 1,962 ug threshold proposed by Plaintiffs. And, as further evidence for the unreliable nature of Plaintiffs' analytical approach, conclusions are reached that are clearly contradicted by a wealth of scientific data. For example, as noted above the liver has consistently been shown to be the most sensitive tissue for the carcinogenic effects of NDMA in laboratory animals. Despite this, Dr. Madigan's analytical approach gives rise to the conclusion that the stomach is a far more sensitive site than the liver, as the lifetime cumulative exposure threshold for NDMA that Dr. Madigan cites for gastric cancer risk is 1,962 ug, whereas that for liver cancer is 14,319 ug.

62. Finally, it is difficult to rationalize the fact that Plaintiffs have selected a single Lifetime Cumulative Exposure level, 1,962 ug, which they identified for a single type of cancer (gastric cancer), as a threshold that would trigger medical monitoring for nine different types of cancer. Notwithstanding the above conclusion that seven of the nine claimed cancer associations with NDMA lack reliable scientific support from dietary epidemiology studies, it is still the case that the Lifetime Cumulative Exposure thresholds for NDMA exposure associated with each of the other cancer sites claimed by Plaintiffs is substantially higher than the threshold identified by Dr. Madigan for gastric cancer. Specifically, even the *lowest* Lifetime Cumulative Exposure thresholds for NDMA exposure claimed by Dr.

Madigan (most, if not all, based on single studies) were: 3,343 ug (Loh: rectal cancer); 4,235 ug (Keszei: esophageal cancer); 4,303 ug (DeStefani: lung cancer); 7,514 ug (Hidajat: bladder, multiple myeloma, prostate, and pancreas cancer); and 14,319 ug (Hidajat: leukemia, lymphoma, and liver cancer). Report of Dr. David Madigan July 7, 2021 ¶ 33-34, Table 1, and § 4. Given that the Lifetime Cumulative Exposure threshold values claimed by Dr. Madigan for other cancer sites range from 1.7-7.3-times *higher* than the Lifetime Cumulative Exposure threshold claimed by Dr. Madigan for gastric cancer, I cannot comprehend the reasoning by which the threshold claimed for gastric cancer should also be applied to cancers of eight other organ sites, each of which exhibited far higher thresholds even by Dr. Madigan's reckoning. This lack of logic is further compounded by the fact that, for gastric cancer and esophageal cancer, the majority of studies listed by Dr. Madigan did not show a statistically significant increased risk of cancer associated with dietary lifetime cumulative exposure to NDMA. Furthermore, the average of significant lifetime cumulative exposure thresholds for lung cancer and colon/colorectal cancer were 11,238 ug and 15,486, respectively, as opposed to the 4,303 and 3,343ug thresholds (respectively) claimed by Dr. Madigan based on single studies. Report of Dr. David Madigan July 7, 2021 ¶ 33-34, Table 1, and § 4.

63. In light of these considerations, I am unable to discern a reasonable – much less a reliable – scientific or medical principle according to which Plaintiffs might have selected 1,962 ug as the Lifetime Cumulative Exposure level for NDMA that should trigger a requirement for medical monitoring. In particular, Plaintiffs' choice to select a threshold for lifetime medical monitoring, 1,962 ug, based upon a single study, for a single cancer type for which the majority of studies evaluated did not show a statistically significant association with dietary lifetime cumulative exposure to NDMA, in preference to the far higher NDMA thresholds (11,238 ug and 15,486 ug) associated with the only two cancers for which at least half of the studies showed a statistically significant association, is irrational, arbitrary, unscientific and lacks a reliable medical basis.

Summary of Opinions

- **Plaintiffs' proposed approach cannot ascertain actual NDMA or NDEA exposures experienced by Plaintiffs.** Plaintiffs fail to articulate a logical medical or scientific basis for their claimed dose/duration/API thresholds for medical monitoring. Moreover, the dose/duration/API thresholds proposed by Plaintiffs cannot be used to determine actual NDMA or NDEA exposures for individual Plaintiffs. For these reasons, and others, Plaintiffs' formulaic claim that "*Plaintiff X consumed a Cumulative Lifetime Threshold of at least XXX units"* [of NDMA, NDEA, or other nitrosamine] and, as a result, "*suffered cellular and genetic injury that creates and/or increases the risk that Plaintiff will develop cancer*" cannot possibly be met by applying the algorithm that Plaintiffs propose.

- **Plaintiffs' claimed dose/duration/API thresholds for medical monitoring for cancer lack a medical or scientific basis.** First, the durations of exposure to valsartan products potentially containing NDMA or NDEA that are proposed by Plaintiffs' algorithm to be sufficient to trigger lifetime medical monitoring (*e.g.*, 108 months, 128 months, 216 months, or 432

months) are nonsensical and untethered to reality, given that the maximum periods of time that valsartan products potentially containing NDMA or NDEA were available on the U.S. market were only 51 months and 75 months, respectively. This fact alone highlights the lack of correspondence between Plaintiffs' proposed algorithm for determining Lifetime Cumulative Exposure thresholds and any reasonable – or even vaguely logical – understanding of the facts regarding potential exposures to NDMA and/or NDEA attributable to ingestion of valsartan products that were even physically possible. Second, Plaintiffs provide no reliable method by which cumulative exposures to NDMA and/or NDEA attributable to ingestion of valsartan products could be ascertained from the proposed (if fatally flawed) dose/duration/API thresholds. In the absence of a reliable method to 'translate' the number of months of exposure to a particular valsartan dose from a particular manufacturer's API to a cumulative exposure to a certain number of micrograms of NDMA and/or NDEA, the body of dietary epidemiology studies referenced by Dr. Madigan (all of which relate cancer risk to the number of micrograms of lifetime cumulative exposure to NDMA and/or NDEA) cannot possibly be used to infer a change in cancer risk among Plaintiffs who ingested valsartan products.

- **Plaintiffs' claimed dose/duration/API thresholds for medical monitoring for cancer are orders of magnitude lower than levels of NDMA, NDEA and other nitrosamines to which human beings are routinely exposed.** Plaintiffs' claim that exposure to as little as 1,962 ug of NDMA, or 624 ug of NDEA, through ingestion of valsartan products is sufficient to warrant lifetime medical monitoring for the development of cancer is illogical and unsupported by medical science, given the facts that these levels of exposure are: (a) lower than the lifetime cumulative exposure to NDMA that FDA considers to be safe; (b) lower than the lifetime cumulative exposure to NDEA that FDA considers to be safe; (c) 2.1 – 10.0-times lower than dietary exposures to preformed NDMA; (d) 4.6 – 14.6-times lower than exposures to NDMA in food, air and water; and (e) >13,000-times lower than estimates of cumulative lifetime exposure to NDMA due to endogenous production. That is, the level of NDMA exposure through ingestion of valsartan containing products claimed by Plaintiffs to be sufficient to warrant lifetime medical monitoring is more than four orders of magnitude lower than the levels of NDMA to which all human beings are exposed as a consequence of normal physiology. If all human beings are exposed to levels of NDMA that are many thousands of times higher than the threshold for medical monitoring proposed by Plaintiffs, such a threshold fails the test of common sense.

- **Even the maximum hypothetical exposures to NDMA and NDEA as a consequence of ingesting valsartan containing products do not cause cancer.** The maximum theoretical total exposure to NDMA to which a Plaintiff could conceivably have been exposed due to ingestion of valsartan products is approximately 2,280-fold *lower* than the lowest dose of NDMA shown to cause a detectable increase in cancer in rats when administered over a lifetime. Moreover, Plaintiffs' apparent claim that exposure to as little as 1,962 ug of NDMA through ingestion of valsartan products is sufficient to warrant lifetime medical monitoring for cancer is remarkable insofar as this level of NDMA exposure is 31,000-fold lower than the lowest dose of NDMA shown to cause a detectable increase in cancer in the most

NDMA-sensitive tissue in rats. Further, Plaintiffs' claim (for Hetero API) that exposure to as little as 241 ug of NDMA through ingestion of valsartan products is sufficient to warrant lifetime medical monitoring for cancer borders on incomprehensible, given that this level of NDMA exposure is more than 250,000-times *lower* than the lowest dose of NDMA shown to cause a detectable increase in cancer in the most NDMA-sensitive tissue in rats. In an analogous manner, the maximum theoretical cumulative exposure to NDEA to which a Plaintiff could conceivably have been exposed due to ingestion of valsartan products is nearly 9,000-times *lower* than a dose of NDEA that does not cause a detectable increase in cancer in rats when administered over a lifetime. Thus, Plaintiffs' apparent claim that exposure to as little as 624 ug of NDEA through ingestion of valsartan containing products is sufficient to warrant lifetime medical monitoring for cancer is remarkable insofar as this level of NDEA exposure is 31,000-fold *lower* than a dose of NDEA that does not cause any detectable increase in cancer in rats when administered over a lifetime. Consequently, it is apparent, to a reasonable degree of medical and scientific certainty, that exposure to NDMA and/or NDEA in valsartan, at the doses to which Plaintiffs were potentially exposed, and for the durations to which Plaintiffs were potentially exposed, would not cause cancer in human beings. Given that conclusion, it would be illogical to posit that levels of exposure to NDMA and NDEA that are even lower are sufficient to warrant lifetime medical monitoring for the development of cancer, as Plaintiffs have proposed.

- **The basis for Plaintiffs' proposed Lifetime Cumulative Exposure threshold for medical monitoring for cancer is subjective, arbitrary, unscientific and unreliable.** The Lifetime Cumulative Exposure thresholds for NDMA and NDEA that Plaintiffs propose as thresholds for lifetime medical monitoring are primarily based upon dietary epidemiology studies, with supplementation from a single occupational study of occupational inhalation exposures in rubber factory workers. For the many reasons discussed at length in my report of August 2, 2021 and my supplemental report, dietary epidemiology studies are an unreliable basis for identifying an amount of NDMA or NDEA associated with an increased risk of cancer in human beings when ingested, as are occupational epidemiology studies of inhaled nitrosamines. Each of these types of studies is subject to critical limitations that preclude reliable interpretations regarding the potential carcinogenic effects of NDMA and NDEA in human populations. Moreover, Plaintiffs' "cherry picking" of data is internally inconsistent. As outlined in this report, there is no reasonable or reliable scientific or medical principle supporting Plaintiffs' selection of Lifetime Cumulative Exposure levels for NDMA that would trigger a requirement for lifetime medical monitoring. In particular, Plaintiffs' choice to select a threshold for lifetime medical monitoring based upon a cancer type for which the majority of studies Drs. Madigan and Panigrahy evaluated did not show a statistically significant association with dietary lifetime cumulative exposure to NDMA in preference to the far higher NDMA thresholds associated with the only two cancers for which at least half of the studies showed a statistically significant association, is irrational, arbitrary, unscientific and lacks a reliable medical basis.

These are my opinions concerning this matter, and I have a sufficient factual basis and good grounds for my conclusions. They are given to a reasonable degree of scientific and medical certainty, and are based on my training and experience and review of the materials included on Exhibit B. I reserve the right to modify this report as additional information is provided to me, including but not limited to additional medical records and the depositions of Plaintiff's experts which are ongoing.

I may use at trial any exhibits as a summary or in support of all of my opinions including: (1) any of the materials, or excerpts identified in this report and attachments, including the materials considered list; (2) excerpts from scientific articles or learned treatises; (3) demonstrative models; (4) exhibits used by Plaintiffs' experts, or other witnesses; (5) any exhibit used in or identified at any deposition taken in this litigation. If further data becomes available, I will review it and consider whether to modify any portion of these opinions.

Date: January 12, 2022

Lewis A. Chodosh, M.D., Ph.D.

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24. Mirvish SS. Formation of N-nitroso compounds: chemistry, kinetics, and in vivo occurrence. *Toxicol Appl Pharmacol.* 1975;31(3):325-351.
25. Mirvish SS. Role of N-nitroso compounds (NOC) and N-nitrosation in etiology of gastric, esophageal, nasopharyngeal and bladder cancer and contribution to cancer of known exposures to NOC. *Cancer Lett.* 1995;93(1):17-48.
26. Siddiqi M, Tricker AR, Preussmann R. Formation of N-nitroso compounds under simulated gastric conditions from Kashmir foodstuffs. *Cancer Lett.* 1988;39(3):259-265.
27. Zeilmaker MJ, Bakker MI, Schothorst R, Slob W. Risk assessment of N-nitrosodimethylamine formed endogenously after fish-with-vegetable meals. *Toxicol Sci.* 2010;116(1):323-335.
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EXHIBIT A

UNIVERSITY OF PENNSYLVANIA – SCHOOL OF MEDICINE
Curriculum Vitae

Date: May 10, 2021

Lewis A. Chodosh, M.D., Ph.D.

Home Address: 1671 Hunters Circle
West Chester, PA 19380

Office Address: 614 Biomedical Research Building II/III
421 Curie Boulevard
Philadelphia, PA 19104-6160

If you are not a U.S. citizen or holder of a permanent visa, please indicate the type of visa you have:
None (U.S. citizen)

Education:

1977-81	B.S.	Yale University (Molecular Biophysics and Biochemistry)
1981-89	M.D.	Harvard Medical School
1983-88	Ph.D.	Massachusetts Institute of Technology (Biochemistry) Laboratory of Dr. Phillip Sharp

Postgraduate Training and Fellowship Appointments:

1989-90	Intern in Medicine, Massachusetts General Hospital, Boston, MA
1990-91	Resident in Medicine, Massachusetts General Hospital, Boston, MA
1991-94	Clinical Fellow in Endocrinology, Endocrine Division, Massachusetts General Hospital, Boston, MA
1991-94	Clinical Fellow in Medicine, Harvard Medical School, Boston, MA
1992-94	Research Fellow in Endocrinology, Endocrine Division, Massachusetts General Hospital, Boston, MA
1992-94	Research Fellow in Medicine, Harvard Medical School, Boston, MA
1992-94	Postdoctoral Research Fellowship, Department of Genetics, Harvard Medical School, Boston, MA Laboratory of Dr. Philip Leder

Military Service

[none]

Faculty Appointments:

1994-2000	Assistant Professor of Molecular and Cellular Engineering University of Pennsylvania School of Medicine
1994-2000	Assistant Professor of Medicine (secondary) Division of Endocrinology, Diabetes and Metabolism University of Pennsylvania School of Medicine
2000-02	Associate Professor with Tenure Department of Molecular and Cellular Engineering

	University of Pennsylvania School of Medicine
2000-06	Associate Professor with Tenure, Department of Medicine Division of Endocrinology, Diabetes and Metabolism (secondary) University of Pennsylvania School of Medicine
2002-06	Associate Professor with Tenure, Department of Cancer Biology University of Pennsylvania School of Medicine
2002-06	Associate Professor with Tenure Department of Cell & Developmental Biology (secondary) University of Pennsylvania School of Medicine
2006-	Professor, Department of Cancer Biology University of Pennsylvania School of Medicine
2006-	Professor, Department of Medicine (secondary) Division of Endocrinology, Diabetes and Metabolism University of Pennsylvania School of Medicine
2006-	Professor, Department of Cell & Developmental Biology (secondary) University of Pennsylvania School of Medicine
2007-08	Interim Chairman, Department of Cancer Biology University of Pennsylvania School of Medicine
2008-12	J. Samuel Staub, M.D. Endowed Professor University of Pennsylvania School of Medicine
2008-	Chairman, Department of Cancer Biology University of Pennsylvania School of Medicine
2018-	Perelman Professor in Cancer Biology (Endowed Chair) University of Pennsylvania

Hospital and Administrative Appointments:

1994-	Member, Abramson Cancer Center, University of Pennsylvania School of Medicine
1995-	Attending Staff Physician, Hospital of the University of Pennsylvania
2000-	Member, Center for Developmental Biology, University of Pennsylvania School of Medicine
2002-	Associate Investigator and Investigator, The Leonard and Madlyn Abramson Family Cancer Research Institute at the University of Pennsylvania Cancer Center
2002-07	Vice Chair, Department of Cancer Biology University of Pennsylvania School of Medicine
2004-08	Founding Co-Director, University of Pennsylvania Small Animal Imaging Facility
2004-	Director, NCI-UPENN Mouse Models for Human Cancers Consortium
2005-13	Program co-Leader, Breast Cancer Program Abramson Cancer Center
2005-16	Director, Cancer Genetics Abramson Family Cancer Research Institute
2016-	Director, Tumor Biology Abramson Family Cancer Research Institute
2005-10	Director, UPENN DOD Breast Cancer Center of Excellence
2005-	Executive Committee

	Abramson Family Cancer Research Institute
2005-	Executive Committee, Abramson Cancer Center
2006-	Associate Director for Basic Science, Abramson Cancer Center
2007-08	Interim Chairman, Department of Cancer Biology
2008-	Chairman, Department of Cancer Biology
2012-	Co-Director, 2-PREVENT Translational Center of Excellence

Medical Licensure: Massachusetts and Pennsylvania

Specialty Certification:

1992-2002	Diplomate in Internal Medicine, American Board of Internal Medicine
1997-2007	Diplomate in Endocrinology and Metabolism, American Board of Internal Medicine

Awards, Honors and Membership in Honorary Societies:

1977	National Merit Scholar
1977	Cornell Ingenuity in Mathematics and Science Award
1977	Harvard Book Prize
1977	Bausch and Lomb Honorary Science Award
1981	*Summa cum laude*, Yale University
1981	Phi Beta Kappa, Yale University
1981	Distinction in Molecular Biophysics and Biochemistry Yale University
1981	Emerson Tuttle Cup for Distinguished Academic Achievement Yale University
1983-89	Medical Scientist Training Program, Harvard Medical School
1985	Leland Fikes Foundation fellow
1989	Leon Reznick Memorial Prize for Excellence in Research Harvard Medical School
1989	Soma Weiss Research Assembly Award, Harvard Medical School
1989	*Magna cum laude*, Harvard Medical School
1992-94	Merck Research Laboratories MD-PhD Fellowship
1996-99	Charles E. Culpeper Foundation Scholarship in the Medical Sciences
1998-2002	U.S. Army Breast Cancer Research Program Career Development Award
2002	AACR-Sidney Kimmel Symposium for Cancer Research Scholar Award
2002	American Society for Clinical Investigation
2005-10	Director, DOD-UPENN Breast Cancer Center of Excellence
2008	Member, Interurban Clinical Club
2008	Association of American Physicians
2009-12	J. Samuel Staub, M.D. Endowed Professor
2013	Dept of Cancer Biology Award for Excellence in Teaching
2017	National Academy of Medicine

2018 Perelman Professor in Cancer Biology

Memberships in Professional and Scientific Societies:

National Societies:

American Society for Clinical Investigation
Association of American Physicians
National Academy of Medicine
American College of Physicians
American Association for Cancer Research
Endocrine Society
American Association for the Advancement of Science
Society for Developmental Biology
American Society for Microbiology
FASEB

Local Societies:

Massachusetts Medical Society

National Scientific Committees:

Electorate Nominating Committee, American Association for the Advancement of Science (2020 -)

Scientific Committees:

Nurses' Health Study I and II, Harvard Medical School, Boston, MA
External Advisory Committee (1998-2016)
Breast Cancer Program Project Grant, Baylor College of Medicine
External Advisory Committee (2002-2007)
CNGI Center Grant, University of Alabama, Birmingham
External Advisory Committee (2002-2004)
Georgetown Cancer Center, Breast Cancer Program Project Grant
External Advisory Committee (2003-2004)
Fox Chase Cancer Center, Breast Cancer and Environmental Research Center, External Advisory Committee (2003-2007)
Dana Farber/Harvard Cancer Center Scientific Advisory Board (2013-present)

Editorial Positions:

1999-2000	Editorial Board, *Breast Cancer Research*
2001-2012	Editorial Board, *Journal of Mammary Gland Biology and Neoplasia*
2003-2014	Associate Editor, *Cancer Biology & Therapy*
2001-2004	Deputy Editor, *Breast Cancer Research*
2005-2008	Senior Editor, *Breast Cancer Research*
2008-present	Editor-in-Chief, *Breast Cancer Research*
2010-2013	Senior Editor, *Cancer Research*

Ad Hoc Reviewer:
Nature, Cell, Science, New England Journal of Medicine, Nature Genetics, Nature Medicine, Cancer Cell, Science Translational Medicine, Molecular & Cellular Biology, Proceedings of the National Academy of Sciences USA, Lancet,

Cancer Research, Development, Human Molecular Genetics
Developmental Biology, Molecular Endocrinology, Oncogene

Peer Review Panels:

1995, 96, 98	U.S. Army Breast Cancer Research Program -
1997, 98, 2000	Susan G. Komen Breast Cancer Foundation
1998, 99	Concert for the Cure
2000	Massachusetts Dept of Public Health Breast Cancer Research Program
2001, 2004	California Breast Cancer Research Program (Chair, 2004)
2009-2012	Cancer Prevention Research Institute of Texas (CPRIT)
2015-2017	AACR Breast Cancer Research Grants Scientific Review Committee
2018	Stand Up To Cancer Canada Metastatic Breast Cancer Peer Review Committee
2019-2020	AACR Outstanding Investigator Award Selection Committee

Academic Committees at the University of Pennsylvania and Affiliated Hospitals:

1994-1996	Internal Advisory Committee, Institute for Human Gene Therapy
1994-1998	Hematology/Immunology Faculty Search Committee, Institute for Human Gene Therapy
1995-1996	Departmental Review Committee - Department of Pediatrics
1995-	Admissions Committee, Combined Degree Program
1997-1998	Departmental Review Committee, Department of Pathology and Laboratory Medicine
1999-2000	Short Term Experience in Research Advisory Committee
1999	Internal Advisory Committee, Cancer Clinical Epidemiology Training Grant, Clinical Center for Epidemiology and Biostatistics
1999-2000	Review Committee, Graduate Group in Neuroscience
2000	LCME Basic Science Task Force
2000-present	Mentoring Program, Combined Degree Program
2002-03	Chair, Cancer Center Working Group on Animal Imaging
2003-2005	Committee on Academic Freedom and Responsibility
2004-2005	Faculty Search Committee for Chair Department of Radiation Oncology
2004-2006	Faculty Search Committee for Chief, Division of Nuclear Medicine
2007	Faculty Search Committee for Chair, Department of Medicine
2008	Faculty Search Committee for Chief, Division of Hematology/Oncology
2010	Faculty Search Committee for Director, Abramson Cancer Center
2010	Faculty Search Committee for Chair, Department of Pathology and Laboratory Medicine
2010-2011	Chair, Review Committee Department of Radiation Oncology
2011-2012	Faculty Search Committee for Chair, Department of Radiology

2011-2012	Chair, Faculty Search Committee for Director, Penn Institute for Immunology
2015-2016	Faculty Search Committee for Chair, Department of Biophysics and Biochemistry
2016-2017	Chair, Faculty Search Committee for Chair, Department of Systems Pharmacology and Translational Therapeutics
2016	President's Consultative Review Committee on the Reappointment of the Dean of the Perelman School of Medicine
2016-	APAC, University of Pennsylvania
2019-	Celgene, Joint Advisory Committee (Ben Garcia)
2020-	Undergraduate Medical Education Committee Perelman School of Medicine, University of Pennsylvania
2020-	Patent Policy Advisory Working Group University of Pennsylvania

Major Teaching and Clinical Responsibilities at the University of Pennsylvania :

1. Attending Physician, Hospital of the University of Pennsylvania (1992-present)
   Endocrine consult service - one month per year (1996-2005)
2. Supervision of graduate students performing thesis research
   Heather Perry Gardner (1996-00)
   Celina D'Cruz (1996-00)
   Stephen Master (Combined Degree Program) (1995-01)
   Gerald Wertheim (Combined Degree Program) (1998-03)
   Susan Moody (Combined Degree Program) (1999-03)
   Robert Boxer (Combined Degree Program) (1999-03)
   Christopher Sarkisian (1998–04)
   Douglas Stairs (1998–04)
   Thomas Yang (1999-05)
   Collin Blakely (Combined Degree Program) (2002-05)
   Joanne Jang (Combined Degree Program) (2002-06)
   Zhandong Liu (2005-10)
   Dania Daye (Combined Degree Program) (2010-2012)
   Jason Jung (2008-13)
   Samantha Eberle (2007-13)
   Daniel Abravanel (Combined Degree Program) (2009-2013)
   Ania Payne (2010-2014)
   Jason Ruth (2010-2014)
   Lauren Smith Pferdehirt (2010-2015)
   Sam Getchell (2010-2016)
   Matthew Paul (2015-2020)
   Takashi Nakamura (2016-)
   Katherine Huang (2018-)
   Saisai Chen (Combined Degree Program) (2018-)
   Brian Benz (2019-)
   Emily Shea (Combined Degree Program) (2021-)
3. Supervision of postdoctoral trainees
   Man Wang, Ph.D. (1995-96)
   Edward Gunther, M.D. (1997-98; 1999-02)

L. Julie Huber, Ph.D. (1997-01)
Eunkyung A. Kauh, M.D., Ph.D. (1999-05)
Charles Bailey, M.D., Ph.D. (2002-07)
Min Wang, Ph.D. (2002-07)
Xiaoping Yang, Ph.D. (2003-07)
Suzanne Bakewell, Ph.D. (2005-09)
James Alvarez, Ph.D. (2005-09)
Chien-Chung Chen, Ph.D. (2004-09)
Elizabeth Yeh, Ph.D. (2006-13)
Yi Feng, Ph.D. (2006-14)
Ann Vernon-Grey, Ph.D. (2006-14)
Heather Martin, Ph.D. (2007-18)
Beth Chislock, Ph.D. (2014-19)
Brett Ecker, M.D. (2016-18)
Francesco Marino, Ph.D. (2017-20)
Matias Escobar, Ph.D. (2017-20)
Amulya Sreekumar, Ph.D. (2017-)
Adetutu Egunsola, Ph.D. (2018-20)

4. Supervision of undergraduate trainees
   Alexander Golant (2001-02)
   Lilangi Ediwickirema (2002-06)
   Srujan Peddapaidi (2005-09)
   Kristi Chakrabarti (2006-10)
   Sanjee Baksh (2012-14)
   Gabriella Puig (2013-16)
   Joseph Kim (2015-17)
   Aaron Solomon (2016-17)
   Alice Zhou (2018-20)
   John Koga (2019-)
   Michelle Lu (2019-)
5. Supervision of masters students
   Alexander Stoddard (2000-01)
   Congzhou Liu (2004-07)
   Elise Giantomaso (2008-09)
6. Supervision of graduate students in laboratory rotations
   1-2 students per semester
7. Supervision of undergraduate students in laboratory rotations
   1-2 students per year
8. Thesis committee member:
   Dongsheng Duan (Ph.D., 1997)
   Hongbing Zhang (Ph.D., 1998)
   Matthew Waterman (Ph.D., 1998)
   Hongtao Zhang (Ph.D., 1999)
   Christopher Wong (Ph.D., 2001)
   Meredith Unger (Ph.D., 2002; Chair)
   Oana Tomescu (Ph.D., 2002)
   Joanna Sax (Ph.D., 2003)
   Michael Keeley (Ph.D., 2005)
   Andrew Gladden (Ph.D., 2005)

Phillip Le (Ph.D., 2005)
Rebekah O'Donnell (Ph.D., 2005)
Jagruti Patel (Ph.D., 2006)
Monica Buzzai (Chair; Ph.D., 2006)
Dara Ditsworth (Chair, Ph.D., 2008)
Karen Urtishak (Ph.D., 2009)
David Schoppy (Ph.D., 2011)
Derek Oldridge (Chair; Ph.D., 2016)
William Rothwell (Chair; Ph.D., 2017)
Emily Fernandez Garcia (Chair;)
Nicholas Perkins (Ph.D., 2020)
Chi-Yun Wu (Ph.D.,)
Austin Pantel (M.S.T.R., 2020)

9. Preliminary examination committee member
Holly Kurzawa (1995)
Christopher Wong (1996)
Praveen Raju (1996)
Gregory Heuer (1998)
Hongbing Zhang (1996)
Michael Gee (1998)
Elizabeth Higbee (2014)
Heide Norton (2015)
Jessica Hsu (2016)
Sofya Osharovich (2017)
Hannah Richter (2017)
Alex Chan (2020)
Hau Truong (2021)

10. Continuing Medical Education
Sacred Heart Hospital, Allentown PA – 1995
Women's Health Research Conference – 1996, 1997
American College of Physicians, Philadelphia, PA

Teaching Lectures:

1995 Ethics in Biomedical Research (faculty facilitator)
1996 CAMB 605 Mammalian Differentiation and Development
Course co-director (12 classes)
1996 Mammary Development and Breast Cancer Risk
Wistar Institute Cancer Biology Training Course
1997 Molecular Basis of Cancer: Part 1
CAMB 610 Molecular Basis of Gene Therapy (course co-director)
1997 Molecular Basis of Cancer: Part 2
CAMB 610 Molecular Basis of Gene Therapy (course co-director)
1997 Tumor Suppressor Gene Strategies for Gene Therapy
CAMB 610 Molecular Basis of Gene Therapy (course co-director)
1997 Strategies for Cancer Gene Therapy
CAMB 610 Molecular Basis of Gene Therapy (course co-director)
1997 The Molecular Basis of Epidemiological Observations
Center for Clinical Epidemiology Training Course

1998 Ethics in Biomedical Research (faculty facilitator)
1998 Anatomy, Development and Physiology of the Breast
Module 1: Brain, Behavior, Endocrine/Reproduction
1998 Molecular Basis of Cancer: Part 1
CAMB 610 Molecular Basis of Gene Therapy (course co-director)
1998 Molecular Basis of Cancer: Part 2
CAMB 610 Molecular Basis of Gene Therapy (course co-director)
1998 Tumor Suppressor Gene strategies for Gene Therapy
CAMB 610 Molecular Basis of Gene Therapy (course co-director)
1998 Strategies for Cancer Gene Therapy
CAMB 610 Molecular Basis of Gene Therapy (course co-director)
1999 Anatomy, Development and Physiology of the Breast
Module 1: Brain, Behavior, Endocrine/Reproduction
1999 The Molecular Basis of Epidemiological Observations
Center for Clinical Epidemiology Training Course
1999 Molecular Basis of Cancer: Part 1
CAMB 610 Molecular Basis of Gene Therapy (course co-director)
1999 Molecular Basis of Cancer: Part 2
CAMB 610 Molecular Basis of Gene Therapy (course co-director)
1999 Tumor Suppressor Gene strategies for Gene Therapy
CAMB 610 Molecular Basis of Gene Therapy (course co-director)
1999 Strategies for Cancer Gene Therapy
CAMB 610 Molecular Basis of Gene Therapy (course co-director)
1999 Strategies for Cancer Gene Therapy
CAMB 610 Molecular Basis of Gene Therapy (course co-director)
1999 Development and Cancer Susceptibility
CAMB 620 Developmental Biology
2000 Anatomy, Development and Physiology of the Breast
Module 1: Brain, Behavior, Endocrine/Reproduction
2000 Molecular Basis of Cancer: Part 1
CAMB 610 Molecular Basis of Gene Therapy
2000 Molecular Basis of Cancer: Part 2
CAMB 610 Molecular Basis of Gene Therapy
2000 Strategies for Cancer Gene Therapy
CAMB 610 Molecular Basis of Gene Therapy
2001 Breast Cancer Genetics and Genomics
CAMB 512 Cancer Genetics
2001 Molecular Basis of Cancer: Part 1
CAMB 610 Molecular Basis of Gene Therapy
2001 Molecular Basis of Cancer: Part 2
CAMB 610 Molecular Basis of Gene Therapy
2001 Strategies for Cancer Gene Therapy
CAMB 610 Molecular Basis of Gene Therapy
2001 Strategies for Cancer Gene Therapy
CAMB 610 Molecular Basis of Gene Therapy
2002 Mammary Development and Cancer: Part 1
CAMB 665 Cancer and Development
2002 Mammary Development and Cancer: Part 2
CAMB 665 Cancer and Development

2002 Mammary Development and Cancer: Part 3
CAMB 665 Cancer and Development
2002 Breast Cancer
CAMB 512 Cancer Genetics
2002 Cancer Genomics
CAMB 512 Cancer Genetics
2002 Molecular Basis of Cancer: Part 1
CAMB 610 Molecular Basis of Gene Therapy
2002 Molecular Basis of Cancer: Part 2
CAMB 610 Molecular Basis of Gene Therapy
2002 Strategies for Cancer Gene Therapy
CAMB 610 Molecular Basis of Gene Therapy
2002 Strategies for Cancer Gene Therapy
CAMB 610 Molecular Basis of Gene Therapy
2003 Mammary Development and Cancer: Part 1
CAMB 665 Cancer and Development
2003 Mammary Development and Cancer: Part 2
CAMB 665 Cancer and Development
2003 Mammary Development and Cancer: Part 3
CAMB 665 Cancer and Development
2004 Molecular Basis of Cancer: Part 1
CAMB 610 Molecular Basis of Gene Therapy
2004 Molecular Basis of Cancer: Part 2
CAMB 610 Molecular Basis of Gene Therapy
2004 Oncogene Addiction and Targeted Therapies
CAMB 542 Topics in Molecular Medicine
2005 Molecular Basis of Cancer: Part 1
CAMB 610 Molecular Basis of Gene Therapy
2005 Molecular Basis of Cancer: Part 2
CAMB 610 Molecular Basis of Gene Therapy
2005 CML, BCR-ABL, and the Philadelphia Chromosome
FRO 514 Frontiers in Cancer Research
2005 Kinase Inhibition: A Paradigm for Molecularly Targeted Therapies
FRO 514 Frontiers in Cancer Research
2006 Breast Cancer Genetics and Genomics: Part 1
CAMB 512 Cancer Genetics
2006 Breast Cancer Genetics and Genomics: Part 2
CAMB 512 Cancer Genetics
2006 Molecular Basis of Cancer: Part 1
CAMB 610 Molecular Basis of Gene Therapy
2006 Molecular Basis of Cancer: Part 2
CAMB 610 Molecular Basis of Gene Therapy
2006 Oncogene Addiction
FRO 514 Frontiers in Cancer Research (course director)
2006 Molecularly Targeted Therapies
FRO 514 Frontiers in Cancer Research (course director)
2006 Cancer Stem Cells
FRO 514 Frontiers in Cancer Research (course director)
2007 Molecular Basis of Cancer: Part 1

CAMB 610 Molecular Basis of Gene Therapy
2007 Molecular Basis of Cancer: Part 2
CAMB 610 Molecular Basis of Gene Therapy
2007 Oncogene Addiction
FRO 514 Frontiers in Cancer Research (course director)
2007 Molecularly Targeted Therapies
FRO 514 Frontiers in Cancer Research (course director)
2007 Cancer Stem Cells
FRO 514 Frontiers in Cancer Research (course director)
2008 Personalized Medicine, Molecularly Targeted Therapies and Oncogene Addiction
Internal Medicine Core Clerkship
2008 Oncogene Addiction
FRO 514 Frontiers in Cancer Research (course director)
2008 Molecularly Targeted Therapies
FRO 514 Frontiers in Cancer Research (course director)
2008 Cancer Stem Cells
FRO 514 Frontiers in Cancer Research (course director)
2009 Cancer Epidemiology
BIOM 502/Mechanisms of Disease and Therapeutic Intervention
2009 Cancer: Part 1
BIO 015 Biology of Human Disease
2009 Cancer: Part 2
BIO 015 Biology of Human Disease
2009 Cancer: Part 3
BIO 015 Biology of Human Disease
2009 Cancer: Part 4
BIO 015 Biology of Human Disease
2009 Oncogene Addiction
FRO 514 Frontiers in Cancer Research (course director)
2009 Molecularly Targeted Therapies
FRO 514 Frontiers in Cancer Research (course director)
2009 Cancer Stem Cells
FRO 514 Frontiers in Cancer Research (course director)
2010 Cancer Epidemiology
BIOM 502/Mechanisms of Disease and Therapeutic Intervention
2010 Cancer: Part 1
BIO 015 Biology of Human Disease
2010 Cancer: Part 2
BIO 015 Biology of Human Disease
2010 Cancer: Part 3
BIO 015 Biology of Human Disease
2010 Cancer: Part 4
BIO 015 Biology of Human Disease
2010 Oncogene Addiction
FRO 514 Frontiers in Cancer Research (course director)
2010 Molecularly Targeted Therapies
FRO 514 Frontiers in Cancer Research (course director)
2010 Cancer Stem Cells
FRO 514 Frontiers in Cancer Research (course director)

2010 Breast Cancer: Personalized Medicine and Targeted Therapies: Part 1
BIOM 502 Mechanisms of Disease and Clinical Applications
2010 Breast Cancer: Personalized Medicine and Targeted Therapies: Part 2
BIOM 502 Mechanisms of Disease and Clinical Applications
2011 Cancer Epidemiology
BIOM 502/Mechanisms of Disease and Therapeutic Intervention
2011 Cancer: Part 1
BIO 015 Biology of Human Disease
2011 Cancer: Part 2
BIO 015 Biology of Human Disease
2011 Cancer: Part 3
BIO 015 Biology of Human Disease
2011 Cancer: Part 4
BIO 015 Biology of Human Disease
2011 Oncogene Addiction
FRO 514 Frontiers in Cancer Research (course director)
2011 Molecularly Targeted Therapies
FRO 514 Frontiers in Cancer Research (course director)
2011 Cancer Stem Cells
FRO 514 Frontiers in Cancer Research (course director)
2011 Breast Cancer: Personalized Medicine and Targeted Therapies: Part 1
BIOM 502 Mechanisms of Disease and Clinical Applications
2011 Breast Cancer: Personalized Medicine and Targeted Therapies: Part 2
BIOM 502 Mechanisms of Disease and Clinical Applications
2012 Cancer Biology
BIO 121 Molecular Biology of Life
2012 Cancer Epidemiology
BIOM 502/Mechanisms of Disease and Therapeutic Intervention
2012 Cancer: Part 1
BIO 015 Biology of Human Disease
2012 Cancer: Part 2
BIO 015 Biology of Human Disease
2012 Cancer: Part 3
BIO 015 Biology of Human Disease
2012 Cancer: Part 4
BIO 015 Biology of Human Disease
2012 Oncogene Addiction
FRO 514 Frontiers in Cancer Research (course director)
2012 Molecularly Targeted Therapies
FRO 514 Frontiers in Cancer Research (course director)
2012 Cancer Stem Cells
FRO 514 Frontiers in Cancer Research (course director)
2013 Cancer Epidemiology
BIOM 502/Mechanisms of Disease and Therapeutic Intervention
2013 Cancer: Part 1
BIO 015 Biology of Human Disease
2013 Cancer: Part 2
BIO 015 Biology of Human Disease
2013 Cancer: Part 3

BIO 015 Biology of Human Disease
2013 Cancer: Part 4
BIO 015 Biology of Human Disease
2013 Cancer: Part 5
BIO 015 Biology of Human Disease
2013 Oncogene Addiction
FRO 514 Frontiers in Cancer Research (course director)
2013 Molecularly Targeted Therapies
FRO 514 Frontiers in Cancer Research (course director)
2013 Cancer Stem Cells
FRO 514 Frontiers in Cancer Research (course director)
2014 Cancer Epidemiology
BIOM 502/Mechanisms of Disease and Therapeutic Intervention
2014 Cancer: Part 1
BIO 015 Biology of Human Disease
2014 Cancer: Part 2
BIO 015 Biology of Human Disease
2014 Cancer: Part 3
BIO 015 Biology of Human Disease
2014 Cancer: Part 4
BIO 015 Biology of Human Disease
2014 Cancer: Part 5
BIO 015 Biology of Human Disease
2014 Oncogene Addiction
FRO 514 Frontiers in Cancer Research (course director)
2014 Molecularly Targeted Therapies
FRO 514 Frontiers in Cancer Research (course director)
2014 Cancer Stem Cells
FRO 514 Frontiers in Cancer Research (course director)
2015 Cancer Epidemiology
Mechanisms of Disease and Therapeutic Intervention
2015 Cancer: Part 1
BIO 015 Biology of Human Disease
2015 Cancer: Part 2
BIO 015 Biology of Human Disease
2015 Cancer: Part 3
BIO 015 Biology of Human Disease
2015 Cancer: Part 4
BIO 015 Biology of Human Disease
2015 Cancer: Part 5
BIO 015 Biology of Human Disease
2015 Cancer Epidemiology
Medical Student Core Curriculum – Module 1
2015 Cancer Biology, Treatment and Natural History
Medical Student Core Curriculum – Module 1
2015 CAMB 512: Cancer Biology and Genetics
Metastasis
2016 Cancer: Part 1
BIO 015 Biology of Human Disease

2016 Cancer: Part 2
BIO 015 Biology of Human Disease
2016 Cancer: Part 3
BIO 015 Biology of Human Disease
2016 Cancer: Part 4
BIO 015 Biology of Human Disease
2016 Cancer: Part 5
BIO 015 Biology of Human Disease
2016 Cancer Epidemiology
Medical Student Core Curriculum – Module 1
2016 Cancer Biology, Treatment and Natural History
Medical Student Core Curriculum – Module 1
2017 Cancer: Part 1
BIO 015 Biology of Human Disease
2017 Cancer: Part 2
BIO 015 Biology of Human Disease
2017 Cancer: Part 3
BIO 015 Biology of Human Disease
2017 Cancer: Part 4
BIO 015 Biology of Human Disease
2017 Cancer: Part 5
BIO 015 Biology of Human Disease
2017 Cancer Epidemiology
Medical Student Core Curriculum – Module 1
2017 Cancer Biology, Treatment and Natural History
Medical Student Core Curriculum – Module 1
2018 CAMB 512: Cancer Biology and Genetics
Metastasis
2018 Cancer Epidemiology
Medical Student Core Curriculum – Module 1
2018 Cancer Biology, Treatment and Natural History
Medical Student Core Curriculum – Module 1
2019 Tissue Growth and Renewal
Medical Student Core Curriculum – Module 1
2019 Cell Cycle Control
Medical Student Core Curriculum – Module 1
2019 Director, Cancer Biology Module
Medical Student Core Curriculum – Module 1
2019 Cancer Epidemiology
Medical Student Core Curriculum – Module 1
2019 Cancer Biology, Treatment and Natural History
Medical Student Core Curriculum – Module 1
2019 Cancer Cell Hallmarks, Heterogeneity and Metastasis
Medical Student Core Curriculum – Module 1
2019 Oncogenic Signaling Pathways: RAS and PI3K
Medical Student Core Curriculum – Module 1
2019 Cancer Cell Metabolism
Medical Student Core Curriculum – Module 1
2019 Molecular Basis of Targeted Therapies

Medical Student Core Curriculum – Module 1
2019 Other Hallmarks of Cancer and Role of Stromal Cells
Medical Student Core Curriculum – Module 1
2019 Cancer Biology, Treatment and Natural History
Medical Student Core Curriculum – Module 1
2020 Tissue Growth and Renewal
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2020 Molecular Basis of Targeted Therapies
Medical Student Core Curriculum – Module 1
2020 Other Hallmarks of Cancer and Role of Stromal Cells
Medical Student Core Curriculum – Module 1

Lectures by Invitation (past 9 years):

August, 2011 "Mechanisms of Breast Cancer Recurrence"
DOD Era of Hope, Orlando, FL
August, 2011 "Minimal Residual Disease and Mammary Stem Cells"
DOD Era of Hope, Orlando, FL
October, 2011 "Rewriting Cancer History"
TEDx Penn 2011, Philadelphia, PA
November, 2011 "Probing Tumor Dormancy and Recurrence"
American Physician Scientists Association 2011 Regional Meeting
Philadelphia, PA
November, 2012 "Modeling Breast Cancer Progression" Dana Farber Cancer Institute
Harvard Medical School, Boston, MA
December, 2012 "Mechanisms of Breast Cancer Dormancy and Recurrence"
San Antonio Breast Cancer Symposium, San Antonio, CA
December, 2012 "Molecular Imaging to Characterize Breast Cancer Models"
San Antonio Breast Cancer Symposium, San Antonio, CA
January, 2013 "Pathways in Tumor Dormancy and Recurrence" AACR Tumor Invasion and Metastasis Special Conference, San Diego, CA
February, 2013 "Mechanisms of Breast Cancer Recurrence" UMDNJ, Newark, NJ
March, 2013 "Mechanisms of Breast Cancer Progression" Duke University, Durham, NC

March, 2013	"Pathways in Tumor Dormancy and Recurrence" Medical University of South Carolina, Charleston, SC
April, 2013	"Mechanisms of Tumor Dormancy and Recurrence" AACR Annual Meeting, Washington, DC
June, 2013	"Breast Cancer Recurrence via Escape from Par-4-dependent Multinucleation and Arrest" Mammary Gland Biology Gordon Research Conference, Stowe, VT
September, 2013	"Dormant Tumor Cell Survival and Recurrence" International Symposium on Minimal Residual Disease, Paris, France
October, 2013	"Mechanisms of Breast Cancer Dormancy and Recurrence" AACR Advances in Breast Cancer Research, San Diego, CA
October, 2013	"Minimal Residual Disease and Breast Cancer Recurrence" Breast Cancer Research Foundation, New York, NY
November, 2013	"Mechanisms of Tumor Dormancy and Recurrence" Massachusetts General Hospital, Harvard Medical School, Boston, MA
December, 2013	"Basic Science: The Year in Review" San Antonio Breast Cancer Symposium, San Antonio, CA
February, 2014	"Tumor Initiating Cells and Minimal Residual Disease" Keystone Symposium on Stem Cells and Cancer, Banff, Alberta
June, 2014	"Probing Tumor Dormancy and Recurrence" NCI Mouse Models for Human Cancers Consortium, Gaithersburg, MD
November, 2014	"Tumor Dormancy and Recurrence: Novel Biology and Therapeutic Opportunities" GlaxoSmithKline Delaware Valley Biology Day, Collegeville, PA
November, 2014	"Tumor Dormancy and Breast Cancer Recurrence" Vanderbilt-Ingram Cancer Center, Nashville, TN
January, 2015	"Tumor Dormancy and Recurrence" Breast Cancer Symposium, George Town, Cayman Islands
April, 2015	"Tumor Dormancy and Recurrence" AACR Annual Meeting, Philadelphia, PA
August, 2015	Keynote speaker 11th Annual Breast Cancer Research and Education Program, Baylor College of Medicine, Montgomery, TX
October, 2015	"Tumor Dormancy and Recurrence" Fourth International AACR Conference on Frontiers in Basic Cancer Research, Philadelphia, PA
March, 2016	"Impact of Obesity, Exercise and Caloric Restriction on Breast Cancer Recurrence in Mice" Transdisciplinary Research on Energetics and Cancer, National Cancer Institute, Rockville, MD
April, 2016	"Tumor Dormancy and Recurrence: Novel Biology and Therapeutic Opportunities" MD Anderson Cancer Center, Houston, TX
November, 2016	"Breast Cancer Recurrence: When the Dragon Awakes" 2016 Jensen Symposium on Breast Cancer University of Cincinnati, Cincinnati, OH
June, 2017	"Tumor Dormancy as a Window to Prevent Metastatic Recurrence" Gordon Research Conference on Mammary Gland Biology Stowe, VT
October, 2017	"Tumor Dormancy and Breast Cancer Recurrence" University of Pittsburgh, Pittsburgh, PA
October, 2017	"Tumor Dormancy and Breast Cancer Recurrence" Symposium on Biology of Cancer: Microenvironment and Metastasis"

	Cold Spring Harbor Laboratory, Cold Spring Harbor, NY
October, 2017	"Breast Cancer Recurrence: Slaying the Sleeping Dragon" Carol M. Baldwin Distinguished Lecture SUNY Upstate Medical University, Syracuse, NY
October, 2017	"Breast Cancer Recurrence: When the Dragon Awakes" Symposium on Metastatic Breast Cancer Jayne Koskinas Ted Giovanis Foundation for Health and Policy Bethesda, MD
May, 2018	"Targeting Tumor Dormancy to Prevent Breast Cancer Recurrence" 11th International Symposium on Minimal Residual Cancer Montpelier, France
November, 2018	"Tumor Dormancy and Metastatic Breast Cancer Recurrence" 5th Annual Metastatic Breast Cancer Conference Johns Hopkins University, Baltimore, MD
December, 2018	"Tumor Dormancy and Cancer Recurrence" Salk Institute Cancer Symposium Salk Institute, La Jolla, CA
December, 2018	"Tumor Dormancy and Late Recurrence" San Antonio Breast Cancer Symposium 2018 San Antonio, TX
April, 2019	"Therapeutic Opportunities: Minimal Residual Disease, Tumor Dormancy and Cancer Recurrence" AACR Annual Meeting, Atlanta, GA
June, 2019	"Minimal Residual Disease, Tumor Dormancy and Cancer Recurrence: Therapeutic Opportunities" Keynote Speaker, Annual Research Symposium, Karmanos Cancer Institute, Wayne State University, Detroit, MI
July, 2019	"Tumor Dormancy and Metastatic Breast Cancer Recurrence" 34th Aspen Cancer Conference, Aspen, CO
October, 2019	"Tumor Cell Dormancy and Breast Cancer Recurrence" British Association for Cancer Research, Newcastle Gateshead, UK
January, 2020	"Tumor Cell Dormancy, Minimal Residual Disease and Breast Cancer Recurrence", Department of Medicine, Department Pediatrics, Division of Hematology/Oncology, Herman B. Wells Center for Pediatric Research, and Simon Cancer Center, Indiana University, Indianapolis, IN
January, 2020	"When the Dragon Awakes: Preventing Breast Cancer Recurrence" Grand Rounds, Simon Cancer Center, Indiana University, Indianapolis, IN
March, 2020	"Minimal Residual Disease and Breast Cancer Recurrence" Cold Spring Harbor Laboratory, Cold Spring Harbor, NY [cancelled due to COVID-19]
July, 2020	"Detecting and Targeting Dormant Tumor Cells to Prevent Death from Recurrent Cancer" Emerson Collective Virtual Symposium
October, 2020	"Changing the Paradigm to Prevent Breast Cancer Recurrence" Yale Alumni Health Network Virtual Symposium
December, 2020	"Dormancy" San Antonio Breast Cancer Symposium Educational Session

Organizing Roles in Scientific Meetings:

April, 2001	Co-Chair, NCI Workshop on BRCA1 Function National Cancer Institute, Bethesda, MD
October, 2003	Conference Co-Chair, AACR Special Conference in Cancer Research: Advances in Breast Cancer Research, Huntington Beach, CA
June, 2004	Chair, Scientific Symposium: "Preclinical Models and Molecular Therapeutics in Cancer" - 2004 ASCO Annual Meeting, New Orleans, LA
April, 2005	"Stem Cells, Metastasis, and Residual Disease: Integrating New Findings in Breast Cancer Research" - Symposium Co-Chair, AACR 96th Annual Meeting, Anaheim, CA
September, 2005	Conference Co-Chair, AACR Special Conference in Cancer Research: Advances in Breast Cancer Research: Genetics, Biology, and Clinical Applications, La Jolla, CA
April, 2006	Program Committee, Chair: Animal Models Subsection AACR 97th Annual Meeting, Washington, DC
October, 2007	Conference Co-Chair, AACR Special Conference in Cancer Research: Advances in Breast Cancer Research: Genetics, Biology, and Clinical Applications, San Diego, CA
December, 2008	SABCS Program Planning Committee, 2008 AACR-CRTC San Antonio Breast Cancer Meeting
October, 2009	Conference Co-Chair, AACR Special Conference in Cancer Research: Advances in Breast Cancer Research: Genetics, Biology, and Clinical Applications, San Diego, CA
November, 2012	NCI Breast Cancer Models Summit, University of Pennsylvania, Philadelphia, PA

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Contribution to peer-reviewed clinical research publications, participation cited but not by authorship:

[none]

Research Publications, non-peer reviewed:

[none]

Abstracts:

Editorials, Reviews, Chapters, including participation in committee reports (print or other media):

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Books:

Mouse Models of Cancer: A Laboratory Manual. Cold Spring Harbor Laboratory Press, Cold Spring Harbor, NY. Abate-Shen C, Politi K, Chodosh LA and Olive KP, eds. 2014.

Alternative Media:

[none]

Patents:

Chodosh, LA: U.S. entitled: "Hormonally Up-Regulated, Neu-Tumor-Associated Kinase". Patent Nos.: 7,119,185 and 7,368,113.

Chodosh, LA: U.S. entitled: "Pregnancy, Up-Regulated Non-Ubiquitous CaM Kinase". Patent Nos.: 7,041,495 and 7,741,111.

Extramural Funding:

ACTIVE

2 R01 CA098371 (Chodosh, PI) 4/18/03 – 2/28/22
NIH/NCI $207,500 annual direct costs
In vivo oncogene-induced tumorigenesis and escape

2 R01 CA148774 (Chodosh, PI) 09/22/10-11/30/22
NIH/NCI $228,750 annual direct costs
Survival and recurrence of dormant cancer cells

2 R01 CA143296 (Chodosh, PI) 06/30/10-05/31/23
NIH/NCI $237,500 annual direct costs
Minimal residual disease and mechanisms of breast cancer recurrence

R01 CA208273 (Chodosh, PI; DeMichele, PI) 12/13/16-11/30/21
NIH/NCI $294,863 annual direct costs
Secondary prevention through surveillance and intervention

R01 CA223816 (Chodosh, PI; Kontos, PI) 07/03/18-5/31/23
NIH/NCI $282,418 annual direct costs
Radiogenomic biomarkers of breast cancer recurrence

W81XWH-17-1-0594 (Chodosh PI) 9/30/17-8/31/20
DOD $264,498 annual direct costs
Dynamic response of disseminated tumor cells and circulating tumor markers to targeted adjuvant therapy

BCRF-16-026 (Chodosh, PI) 10/01/04-9/30/21
Breast Cancer Research Foundation $145,833 annual direct costs
Pathways in breast cancer progression

W81XWH-20-1-0008 (Chodosh PI; DeMichele Partnering PI) 1/15/20-1/14/22
DOD $652,188 annual direct costs
Detecting and defining residual tumor cell subsets to enable recurrence prevention in early stage breast cancer patients

P30 CA016520 (Vonderheide) 12/01/10-11/30/20
NIH/NCI $30,890
University of Pennsylvania Cancer Center Core Grant

EXHIBIT B

GT GreenbergTraurig

In re: Valsartan, Losartan, and Irbesartan Products Liability Litigation
Case No. 19-2875

LEWIS A. CHODOSH, M.D., PH.D.
AMENDED LIST OF MATERIALS CONSIDERED
JANUARY 12, 2021

MATERIALS CONSIDERED	BATES NOS.
MDL PLEADINGS AND GENERAL DOCUMENTS	
Am. Master Personal Injury Complaint	N/A
2019.06.17 Am. Master Medical Monitoring Complaint	N/A
2021.11.01 – Consolidated Third Amended Medical Monitoring Complaint	N/A
2021.02.11 Letter from Lori G. Cohen to Judge Vanaskie	N/A
2021.02.11 Letter from Adam Slater Providing an Overview	N/A
Am. Master Economic Loss Complaint	N/A
Memorandum ISO of the Medical Monitoring Plaintiffs' Motion for Class Certification with Exhibits	N/A
REPORTS AND DISCLOSURES	
2021.07.04 Dr. Mahyar Etiminan Report • CV	N/A
2021.07.06 Dr. Stephen Hecht Report • CV • List of Documents Reviewed • Literature Referenced	N/A
2021.11.10 Dr. Edward H. Kaplan Report • CV • List of Materials Considered	N/A
2021.07.06 Dr. Stephen Lagana Report • CV	N/A
2021.07.07 Dr. David Madigan Report • CV	N/A
2021.07.06 Dr. Dipak Panigrahy Report • CV	N/A
2021.11.10 Dr. Zirui Song Report • CV • Materials Relied On	N/A
2021.05.18 Dr. Nagi Kumar Report • CV	N/A
2021.05.30 Dr. Steven Bird Report • CV	N/A

2021.08.02 – Report of Michael Bottorff	N/A
2021.08.02 – Report of Janice Britt	N/A
2021.08.02 – Report of Daniel Catenacci	N/A
2021.08.02 – Report of Lewis Chodosh	N/A
2021.08.02 – Report of Jon Fryzek	N/A
2021.08.02 – Report of John Gibb	N/A
2021.08.02 – Report of George Johnson	N/A
2021.08.02 – Report of Lee-Jen Wei	N/A
WRITTEN DISCOVERY	
Plaintiff's Disclosure of Cancer Types	N/A
TEVA DOCUMENTS	
2018.07.06 Teva Health Hazard Assessment re Valsartan	TEVA-MDL2875-00274341
2018.07.06 Teva Health Hazard Assessment re Valsartan/HCTZ	TEVA-MDL2875-00274351
2018.07.10 Health Hazard Assessment of Amlodipine Valsartan	TEVA-MDL2875-00680243
2018.07.10 Health Hazard Assessment of Amlodipine Valsartan HCTZ	TEVA-MDL2875-00680244
2018.06.29 Teva Toxicological Assessment of NDMA impurity in valsartan by Dr. Nudelman	TEVA-MDL2875-00274358
2018.11.12 Tox Assessment for NDEA in Valsartan by Dr. Nudelman	TEVA-MDL-00953115
2019.03.13 Tox Assessment for NDMA and NDEA in Sartan Drugs in Parellel	TEVA-MDL2875-00773542
2019.07.03 Teva Risk Assessment Report for Valsartan Huahai	TEVA-MDL-00693424
2019.07.03 Teva Risk Assessment Report for Valsartan Mylan	TEVA-MDL-00693422
2019.07.18 Teva Valsartan Analytical Drug Substance & Drug Product Testing Results	TEVA-MDL-0063060
ZHP root cause	TEVA-MDL2875-00783229
Mylan root cause	TEVA-MDL2875-00019995
CBE-30 for ANDA 091519 – Valsartan/HCTZ w/ ZHP API	TEVA-MDL2875-00001886
CBE-30 for ANDA 090642 – Valsartan w/ ZHP API	TEVA-MDL2875-00013107
sANDA Approval by FDA for ANDA 091519	TEVA-MDL2875-00133642
sANDA Approval by FDA for ANDA 090642	TEVA-MDL2875-00354034
Valsartan sales	TEVA-MDL2875-00019951
Valsartan sales	TEVA-MDL2875-00019954
Email with test results	TEVA-MDL2875-00546489
Response to FDA Request for Information (RFI) for Valsartan (Jan 30, 2019)	TEVA-MDL2875-00546490
Testing result of NDMA in valsartan	TEVA-MDL2875-00546493
Balkanpharma Oupnitsa results for NOMA content in Valsartan API, manufactured by Zhejiang Huahai Co.,Ltd	TEVA-MDL2875-00546494

Balkanpharma Oupnitsa results for NOMA content in Valsartan API, manufactured by Zhejiang Huahai Co.,Ltd	TEVA-MDL2875-00546495
Miscellaneous Study Report	TEVA-MDL2875-00546496
Bafkanpharma Dupnitsa results for NOMA content in Valsartan tabfets and Valsartan/HCT tablets	TEVA-MDL2875-00546511
Toxicological Qualification of Impurity in Amlodipine/Valsartan Tablets	TEVA-MDL2875-00158435
Toxicological Qualification of Impurity in Amlodipine/Valsartan Tablets	TEVA-MDL2875-00158435
Computational Mutagenicity Report for Potential Impurity in Valsartan	TEVA-MDL2875-0025993
Toxicological Qualification of [] EP Impurity D in [], Valsartan, and Hydrochlorothiazide Tables	TEVA-MDL2875-00260014
Computational Mutagenicity and Control Recommendations for Potential Impurities in Valsartan	TEVA-MDL2875-00260232
Computational Mutagenicity and Control Recommendations for Potential Impurities in Valsartan	TEVA-MDL2875-00260411
Computational Mutagenicity and Control Recommendations for Potential Impurities in Valsartan	TEVA-MDL2875-00351418
Health Hazard Assessment, Valsartan Containing Products	TEVA-MDL2875-00426004
1.11.1 Response to FDA Request for Information – Quality ANDA# 091235	TEVA-MDL2875-00539061
1.11.1 Response to FDA Request for Information – Quality ANDA# 200435	TEVA-MDL2875-00539082
Valsartan Quality Reports	TEVA-MDL2875-00693421
Global Quality Report # 2019-GQ-005 1v1	TEVA-MDL2875-00693423
Global Quality Report # 2019-GQ-005 1v1	TEVA-MDL2875-00765603
1.11.1 Response to FDA Request for Information – Quality ANDA # 091519	TEVA-MDL2875-00765609
Health Hazard Assessment, Valsartan and Hydrochlorothiazide Tablets	TEVA-MDL2875-00436691
Health Hazard Assessment, Valsartan and Hydrochlorothiazide Tablets	TEVA-MDL2875-00436689
FDA/REGULATORY GUIDANCES AND DOCUMENTS	
Publicly Available FDA Documents	
2018.07.13 FDA Announces Voluntary Recall, FDA News Release	N/A
2018.07.17 Teva Issues Voluntary Recall	N/A
2018.07.18 Recalled US Valsartan Labels	N/A
2018.08.30 FDA Statement on Ongoing Investigation into Valsartan Impurities	N/A
2018.11.27 Teva Announces Voluntary Recall of All Amlodipine	N/A
2019.04.04 FDA Statement – Update on Recall	N/A

2019.04.15 Laboratory analysis of valsartan products	N/A
2019.06.13 Valisure Citizens Petition	N/A
2020.10.02 FDA Overview of Guidance for Industry	N/A
2020.12.04 - Laboratory analysis of valsartan products - FDA	N/A
2021 - Laboratory analysis of valsartan products, https://www.fda.gov/drugs/drug-safety-and-availability/laboratory-analysis-valsartan-products	N/A
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DEPOSITION TRANSCRIPTS (WITH EXHIBITS)	
Teva Witnesses	
Raphael Nudelman – 04.08.2021 - Transcript	N/A
79 - Notice of Deposition	N/A
80 - Response to Plaintiffs' Document Requests	N/A
81 - Resumé Raphael Nudelman, Ph.D., ERT	TEVA-MDL2875-DEPS-000019-24
82 - LinkedIn Raphael Nudelman, Ph.D., ERT	TEVA-MDL2875-DEPS-000025-26
83 - No Exhibit	No Exhibit
84 - No Exhibit	No Exhibit
85 - Audit Report On ZHP (Chuannan Site) 9/2/11	TEVA-MDL2875-00051288

86 - No Exhibit	No Exhibit
87 - Auditing of API Manufacturers 3/27/12	TEVA-MDL2875-00102747
88 - E-mail, 7/4/12 Subject, Genotoxicity Evaluation for Valsartan (Azide Route)	TEVA-MDL2875-00539802
89 - Computational Toxicology Report for Valsartan Reagents and Intermediates 7/19/12	TEVA-MDL2875-00259857
90 - E-mail Thread 8/1/12 Subject, Acetaldehyde Toxicity	TEVA-MDL2875-00514864-66
91 - No Exhibit	No Exhibit
92 - No Exhibit	No Exhibit
93 - E-mail Thread 3/30/14 Subject, Amlodipine Besilate	TEVA-MDL2875-00158436-39
94 - Caspofungin 50-70 mg Powder for Concentrate For Solution for Infusion 3.2.P.2	TEVA-MDL2875-00917440-19
95 - Computational Mutagenicity Report For Potential Impurity In Valsartan 10/14/15	TEVA-MDL2875-00259986-87
96 - E-mail Thread 6/15/16 Subject, Toxicology of Hydrolyzed Calpronium Chloride	TEVA-MDL2875-00158463-69
97 - No Exhibit	No Exhibit
98 - E-mail Thread 12/19/17 Subject, Valsartan Proposal for API Specifications Revision US	TEVA-MDL2875-00082321-24
99 - Computational Mutagenicity and Control Recommendations For Potential Impurities In Valsartan	TEVA-MDL2875-00158698-05
100 - Quality Risk Management for Cross Contamination Control	TEVA-MDL2875-00260122
101 - E-mail Thread 6/28/18 Urgent and Important Genotoxic Impurity Notification	TEVA-MDL2875-00056559-61
102 - Request for Safety Assessment of NDMA for Valsartan Dose 1x Daily for 320mg, 160mg, 80mg (June 28, 2018)	TEVA-MDL2875-00425812-14
103 - Toxicological Assessment for NDMA In Valsartan Drug Substance 6/29/18	TEVA-MDL2875-00158529
104 - No Exhibit	No Exhibit
105 - E-mail Thread 7/3/18 Subject, Urgent Valsartan Safety Assessment Request	TEVA-MDL2875-00158519-22
106 - E-mail Thread 7/4/18 Subject Valsartan Urgent	TEVA-MDL2875-00056924-29
107 - E-mail Thread 7/4/18 Subject, Valsartan Urgent	TEVA-MDL2875-00514896-02
108 - E-mail Thread 7/4/18 Subject, Valsartan Urgent	TEVA-MDL2875-00020609-18
109 - E-mail Thread 7/5/18 Subject, Valsartan Urgent	TEVA-MDL2875-00158540-41
110 - E-mail Thread 7/5/18 Subject, Valsartan Urgent	TEVA-MDL2875-00495085
111 - E-mail Thread 7/5/18 Subject, Valsartan HHA draft V3 for Review	TEVA-MDL2875-00057083-85
112 - No Exhibit	No Exhibit
113 - No Exhibit	No Exhibit
114 - Draft HHA Valsartan Tablets 40, 80, 160, 320 Multiple Lots	TEVA-MDL2875-00057086-94

115 - No Exhibit	No Exhibit
116 - E-mail Thread 7/12/18 Subject, Valsartan	TEVA-MDL2875-00158544
117 - E-mail Thread 7/13/18 Subject, Valsartan Request from Hong Kong	TEVA-MDL2875-00540426-30
118 - E-mail Thread 7/13/18 Subject, EDQM Valsartan Provisional Limit Potential Impact on HHAs	TEVA-MDL2875-00021077-78
119 - Certification of Substances Department 8/10/18 Request for Information Relating to EU Referral Article 31 of Directive 2001/83/EC	N/A
120 - No Exhibit	No Exhibit
121 - E-mail Thread 9/6/18 Subject, Draft Valsartan Letter	TEVA-MDL2875-00552854-59
121A - NDMA Acceptable Limit (Handwritten Document From Plaintiff's Counsel Watt)	N/A
122 - E-mail Thread 10/22/18 Subject, NDMA & NDEA Limits	TEVA-MDL2875-00514942-43
123 - No Exhibit	No Exhibit
124 - E-mail Thread 12/27/18 Subject, Update 26th December 2018 RV Update	TEVA-MDL2875-00540783-88
125 - No Exhibit	No Exhibit
126 - E-mail Thread 3/26/19 Subject, Snodin & Elder Commentary	TEVA-MDL2875-00492386
127 - E-mail Thread 6/27/19 Subject, Request to be An Honorable Keynote Speaker	TEVA-MDL2875-00540844-46
128 - E-mail Thread 8/11/19 Subject, Editor's Spotlight Switzerland	TEVA-MDL2875-00158591-93
129 - Questionnaire for Excipient Nitrosamines Risk Evaluation	TEVA-MDL2875-00158603-09
130 - E-mail Thread 10/23/19 Subject, Ranitidine NDMA Formation	TEVA-MDL2875-00562588-97
131 - No Exhibit	No Exhibit
132 - No Exhibit	No Exhibit
133 - HHA Valsartan Tablets 40, 60,160, 320mg Multiple Lots	TEVA-MDL2875-00274341-49
134 - Toxicological Assessment for NDMA And NDEA in Parallel in Sartan-Drug Substances	TEVA-MDL2875-00773542
Expert Witnesses	
2021.09.16 –Transcript of Dr. Michael Bottorff • Exhibits 1-8	N/A
2021.09.13 –Transcript of Dr. Daniel Catenacci **2021.09.14 – Transcript of Dr. Daniel Catenacci (rough)** • Exhibits 1-12	N/A

2021.08.24 –Transcript of Mayur Etminan • Exhibits 1-30	N/A
2021.08.17 – Transcript of Dr. Steven Hecht • Exhibits 1-32	N/A
2021.08.05 – Transcript of Dr. David Madigan • Exhibits 1-26	N/A
2021.08.13 – Transcript of Dr. Stephen Lagana • Exhibits 1-33	N/A
2021.09.10 and 2021.09.11 – Transcripts of Dr. Dipak Panigrahy • Exhibits 1-29	N/A
2021.09.14 and 2021.09.15 –Transcripts of Lee-Jen Wei • Exhibits 1-15	N/A
BELLWETHER PLAINTIFFS	
Bonmon, Yolanda	
Plaintiff Fact Sheet	
Plaintiff Fact Sheets	YBonmon-PFS-000001 – 748
Deposition	
Bonmon, Yolanda – 2021.04.20 – Transcript	N/A
1 – 2021.04.16 Plaintiff Fact Sheet	N/A
2 – 2021.04.16 Signed Declaration of Plaintiff Fact Sheet	N/A
3 – Photograph of Valsartan Bottle	YBonmon-PPR-000319
4 - 2019.06.17 Amended Complaint - Master Personal Injury Complaint	N/A
5 – 2020.07.21 Bonmon Short Form Complaint	N/A
6 – Bonmon Medical Records from Charles K. Embry, MD	YBonmon-CEmbry-000001 – 88
7 – Bonmon Pharmacy Records from Apothecare Pharmacy	YBonmon-ApothPIII-000001 – 13
8 – Bonmon Medical Records from Bluegrass Women's Healthcare	YBonmon-BlueWHC-000001 – 55
9 – Bonmon Medical Records from Central Medical Associates	YBonmon-CMA-000035 – 89
10 – Bonmon Medical Record from UK Healthcare	YBonmon-PPR-000030
11 – Bonmon Medical Records from Central Medical Associates	YBonmon-CMA-000035 – 89
12 – Bonmon Medical Records from Central Medical Associates	YBonmon-CMA-000001 – 34
13 – Bonmon Medical Records from Hardin Memorial Hospital	YBonmon-HMH-MD-000019 – 480
14 – Bonmon Medical Records from Charles K. Embry, MD	YBonmon-CEmbry-000001 – 88

15 – 2021.04.16 Plaintiff Fact Sheet	N/A
16 – Bonmon executed authorization for New Hope Foster Agency	N/A
17 – Bonmon records from New Hope Foster Homes, Inc.	YBonmon-NHFAFC-HR-000001
18 – Bonmon Executed Tax Authorization	N/A
Medical Records	
Plaintiff Produced Records	YBonmon-PPR-000001 – 658
Apothecare Pharmacy III	YBonmon-ApothPIII-000001 – 13
Bluegrass Women's Healthcare	YBonmon-BlueWHC-000001 – 59
Central Medical Associates PLLC	YBonmon-CMA-000001 – 267
Embry Charles, MD	YBonmon-CEmbry-000001 – 88
Hardin Memorial Hospital	YBonmon-HMH-000001 – 521
Laboratory Corporations of America	YBonmon-LCA-000001 – 7
Lincoln Trail Diagnostics	YBonmon-LTD-000001 – 52
Norton Cancer Institute	YBonmon-NCI-000001 – 11
Norton Healthcare	YBonmon-NortonHealthcare-000001 – 559
UK Albert B. Chandler Hospital	YBonmon-UKABCH-000001 – 162
Walgreen Company	YBonmon-WC-000001 – 9
Briones, Joe	
Plaintiff Fact Sheet	
Plaintiff Fact Sheet, 02/02/2021	JBriones-PFS-000095 – 187
Medical Records	
Plaintiff Produced Records	JBriones-PPR-000001 – 441
Citizens Medical Center	JBriones-CMCen-000001 – 407
DeTar Hospital	JBriones-DeTarH-000001 – 436
Envision Pharmacies	JBriones-EnvisionP-000001 – 2
Gastroenterology of Victoria	JBriones-GVictoria-000001 – 7
HEB Pharmacy	JBriones-HEBPharm-000001 – 2
Minocha, Gulshan MD	JBriones-GKMinocha-000001 – 217
Regional Path Assocs	JBriones-RPA-000001 – 2
University of Texas MD Anderson	JBriones-UTMDACC-RD-000001 – 39
University of Texas MD Anderson Cancer Center	JBriones-UTMDACC-000001 – 8520
Dawson, Nellie	
Plaintiff Fact Sheet	
Plaintiff Fact Sheet 05/14/2020	NDawson-PFS-000092-000180
Medical Records	
Plaintiff-Produced Medical Records	NDawson-PPR-000001-000179
3HC Home Health Hospice Healthcare	NDawson-3HCHH-H-HC-000001-000114
Jordan And Assocs Gastroenterology PA	NDawson-J&AG-000001-000068

Riverdale Family Medicine PA	NDawson-RFM-000001-000332
UNC Health Care System Path Dept	NDawson-UNCHCS-PD-000001-000001
UNC HealthCare System Rad Dept	NDawson-UNCHCS-RD-000001-000001
Dufrene, Lana	
Plaintiff Fact Sheet	
Plaintiff Fact Sheets	LDufrene-PFS-000001 – 186
Medical Records	
Plaintiff Produced Records	LDufrene-PPR-000001 – 178
Cardiovascular Institute of the South	LDufrene-CIS-000001 – 183
Lady of the Sea General Hospital	LDufrene-LSGH-000001 – 77
Leonard J. Chabet Medical Center	LDufrene-LJCMC-000001 – 2271
Ochsner Family Doctor Clinic	LDufrene-OFDC-000001 – 193
Racelands Pharmacy	LDufrene-RPE-000001 – 13
Walmart Pharmacy	LDufrene-WMS-000001 – 20
Garcia, Robert	
Plaintiff Fact Sheet	
Plaintiff Fact Sheet, 03/12/2021	RGarcia-PFS-000188 – 283
Medical Records	
Plaintiff Produced Records	RGarcia-PPR-000001 – 434
Baylor St. Lukes Medical Center	RGarcia-BStLMC-000001 – 831
CVS Pharmacy	RGarcia-CVS-000001 – 25
Express Scripts Inc.	RGarcia-ES-000001 – 12
HEB Pharmacy	RGarcia-HEBPharm-000001 – 25
Kelsey Pharmacy Berthelsen	RGarcia-KelseyP-000001 – 3
Kelsey Seybold Clinic	RGarcia-KSC-000001 – 2128
Texas Digestive Disease Consultants	RGarcia-TexasDDC-000001 – 42
Walgreen Company	RGarcia-WC-000001 – 79
Kennedy, Paulette	
Plaintiff Fact Sheet	
Plaintiff Fact Sheet, 06/21/2021	PKennedy-PFS-000377 – 470
Medical Records	
Plaintiff Produced Records	PKennedy-PPR-000001 – 590
Baylor Scott and White Medical Center	PKennedy-BS&WMC-000001 – 438
Dallas Cardiac Associates	PKennedy-DCA-000001 – 25
Dallas Nephrology	PKennedy-DallasNephA-000001 – 125
Kroger Pharmacy	PKennedy-KrogerPharm-000001 – 9
Lajara, Rosemarie, MD	PKennedy-RLajara-000001 – 10
Medical City Dallas	PKennedy-MCDH-000001 – 453
Northstar Diagnostic Imaging	PKennedy-NStarDI-000001 – 33
Solis Mammography	PKennedy-SolisM-000001 – 34

Southern Endocrinology and Diabetes Association	PKennedy-SEndo&DA-000001 – 33
Texas Breast Specialists	PKennedy-TBS-000001 – 168
Texas Colon and Rectal Surgeons	PKennedy-TC&RSurgeons-000001 – 128
Texas Oncology	PKennedy-TOncology-000001 – 385
Walgreen Company	PKennedy-WC-000001 – 91
Kinkela, Silvano	
Plaintiff Fact Sheet	
Plaintiff Fact Sheet, 06/11/2021	SKinkela-PFS-00547 – 641
Medical Records	
Plaintiff Produced Records	SKinkela-PPR-00001 – 430
Aaron, Jay S., MD	SKinkela-JSAaron-00001 – 85
Advance Urology Centers of New York	SKinkela-AUCNY-00001 – 33
East Virginia ENT Specialists	SKinkela-EVEN&TS-00001 – 18
Lackawana County Dermatology Associates	SKinkela-LVDA-00001 – 19
Optum Rx	SKinkela-OptumRx-00001 – 138
Pulmonary And Critical Care Specialists	SKinkela-P&CCS-00001 – 56
Sentara Leigh Hospital	SKinkela-SentaraLH-00001 – 262
Sentara Surgery Specialists	SKinkela-SSS-00001 – 749
Urology Associates of the Poconos	SKinkela-UAP-00001 – 62
Virginia Oncology Associates	SKinkela-VOA-00001 – 93
Walgreen Company	SKinkela-WC-00001 – 24
Lee, Robert	
Plaintiff Fact Sheet	
Plaintiff Fact Sheets	RLee-PFS-000001-000167
Medical Records	
Plaintiff-Produced Medical Records	RLee-PPR-000001-000958
Blue Cross Blue Shield of South Carolina	RLee-BCBSSC-000001-000092
Ctrs for Medicare and Medicaid Svcs Region 4	RLee-CMMS-R4-000001-000126
Death Certificate Proof Of Authority	RLee-DCPOA-000001-000002
Family Healthcare Clinton	RLee-FH-C-000001-000404
Greenville Health System Patient Accts	RLee-GHS-BD-000001-000027
Greenville Health System Med Recs Dept	RLee-GHS-MD-000001-001985
Greenville Memorial Hosp Rad Dept	RLee-GMH-RD-000001-000017
Greenville Memorial Hospital -Billing	RLee-GMH-BD-000001-000005
Ingles Markets, Inc.	RLee-InglesM-000001-000029
Walmart Pharmacy	RLee-WMS-000001-000027
Meeks, Ronald	
Plaintiff Fact Sheet	
Plaintiff Fact Sheets	RMeeks-PFS-000001-000288
Medical Records	
Plaintiff-Produced Medical Records	RMeeks-PPR-000001-006576

Central Arkansas Veterans Healthcare System Med Recs Dept	RMeeks-CAVHS-MD-000001-000011
Central Arkansas Veterans Healthcare System Path Dept	RMeeks-CAVHS-PD-000001-000002
Death Certificate Proof Of Authority	RMeeks-DCPOA-000001-000005
East Jefferson Cardiovascular Specialists Inc Med Recs Dept	RMeeks-EJCS-MD-000001-000163
East Jefferson General Hosp Path Dept	RMeeks-EastJGH-PD-000001-000001
East Jefferson General Hosp Med Recs Dept	RMeeks-EJGH-000751-002405
East Jefferson General Hosp Patient Accts	RMeeks-EastJGH-BD-000001-000027
East Jefferson General Hosp Rad Dept	RMeeks-EastJGH-RD-000001-000001
East Jefferson Internal Medicine	RMeeks-EJIM-000001-000057
Med Plaza ENT Physicians	RMeeks-MPENTP-000001-000036
Nola Discount Pharmacy Pharmacy	RMeeks-NDP-000001-000027
Ochsner Med Ctr Release of Information	RMeeks-OchsnerMC-MD-000001-003194
Ochsner Med Ctr Patient Accts	RMeeks-OchsnerMC-BD-000001-000124
Ochsner Med Ctr Kenner Med Recs Dept	RMeeks-OMC-K-MD-000001-000797
Ochsner Med Ctr Kenner Patient Accts	RMeeks-OMC-K-BD-000001-000010
Ochsner Med Ctr Kenner Path Dept	RMeeks-OMC-K-PD-000001-000001
Ochsner Med Ctr Kenner Rad Dept	RMeeks-OMC-K-RD-000001-000001
Ochsner Medical Complex - NR Cert Ltr	RMeeks-OMComp-000001-000001
Smith Kenneth B MD	RMeeks-KBSmith-000001-000175
Southeast Louisiana Veterans HealthCare System Rad Dept	RMeeks-SLVHCS-RD-000001-000062
Southeast Louisiana Veterans Health Care System	RMeeks-SLVHCS-RD-000008-000009
Tulane Univ Hosp and Clinic Rad Dept	RMeeks-TUHC-RD-000001-000003
Tulane Univ Hosp and Clinic Med Recs Dept	RMeeks-TUHC-MD-000001-000001
Univ Med Ctr New Orleans Rad Dept	RMeeks-UMCNO-RD-000001-000002
Univ Med Ctr New Orleans Patient Accts	RMeeks-UMCNO-BD-000001-000009
Univ Med Ctr New Orleans Path Dept	RMeeks-UMCNO-PD-000001-000001

Univ Med Ctr New Orleans Med Recs Dept	RMeeks-UMCNO-MD-000001-000389
Suits, James	
Plaintiff Fact Sheet	
Plaintiff Fact Sheets	JSuits-PFS-000001-001224
Medical Records	
Plaintiff-Produced Medical Records	JSuits-PPR-000001-001335
Aetna US Healthcare Legal Support Svcs	JSuits-AUSH-000001-000002
John Deere - NRS	JSuits-JohnDeere-HR-000001-000001
McCaysville Internal Medicine	JSuits-McCIM-000001-000251
Mutual of Omaha Insurance Company Claims Dept	JSuits-MOIC-000001-000003
Premier Surgical Assocs Cleveland	JSuits-PremierSAC-000001-000048
Tallent Drug Store	JSuits-TDS-000001-000036
Uhlik, Allen, MD	JSuits-AUhlik-000001-000383
Weygandt, Robert	
Plaintiff Fact Sheet	
Second Amended Plaintiff Fact Sheet, 04/07/20	RWeygandt-PFS-000001-000090
Depositions	
Weygandt, Martha – 2021.04.13 – Transcript	N/A
1 – 2021.03.08 Plaintiff Fact Sheet	N/A
2 – 2021.03.08 Signed Declaration of Plaintiff Fact Sheet	N/A
3 – 2014.05.22 Bankruptcy Motion for Expedited hearing on Motion to Use Cash Collateral	N/A
4 - 2019.06.17 Amended Complaint - Master Personal Injury Complaint	N/A
5 – 2020.10.21 Weygandt Short Form Complaint	N/A
6 – Weygandt Medical Records produced by Plaintiff	Various
7 – Robert Weygandt Death Certificate	RWeygandt-DCPOA-000001
8 – Weygandt Medical Records produced by Plaintiff	Various
9 – Weygandt Safeway Insurance Records	RWeygandt-Safeway-00003 – 17
10 – Weygandt Medical Record from Endocrine Associates of Dallas	RWeygandt-EAD-000055 – 60
11 – Weygandt Medical Records from Carrell Clinic	RWeygandt-CarrellC-00003 – 6
12 – Weygandt Medical Records from Dr. Potluri	RWeygandt-HHBP-MD000431 – 433
13 – Weygandt Medical Record from Endocrine Associates of Dallas	RWeygandt-EAD-000074 – 78
14 – Weygandt Medical Record from Endocrine Associates of Dallas	N/A
15 – Weygandt Medical Record from Endocrine Associates of Dallas	N/A
16 – 2020.10.30 Albertson's Defendant Fact Sheet	N/A
Medical Records	

Plaintiff-Produced Medical Records	RWeygandt-PPR-001547-001817
Abrams Royal Pharmacy II Pharmacy	RWeygandt-ARPharmII-000001-000002
Advanced Imaging Center	RWeygandt-AImagingCe-000001-83
Aetna US Healthcare Legal Support Svcs	RWeygandt-AUSH-000001-000013
Baylor Regional Med Ctr at Plano Med Recs Dept	RWeygandt-BRMCP-MD-000001-000307
Baylor Regional Med Ctr at Plano Path Dept	RWeygandt-BRMCP-PD-000001-000001
Baylor Scott and White Health Rad Dept	RWeygandt-BSW-RD-000001-000002
Baylor Scott and White Health - NRS	RWeygandt-BSW-PD-000001-000001
Baylor Scott and White Health Med Recs Dept	RWeygandt-BSW-MD-000001-000002
Baylor Scott and White Health	RWeygandt-BSW-BD-000001-17
Baylor Surgicare at Plano Patient Accts	RWeygandt-BSPlano-BD-000001-000002
Baylor Surgicare at Plano - NR Radiology Cert	RWeygandt-BSPlano-RD-000001
Blue Cross Blue Shield of Texas Claims Dept	RWeygandt-BCBST-000001-000048
Carrell Clinic - Medical	RWeygandt-CarrellC-000001-000057
Clinical Path Labs Inc	RWeygandt-CPL-000001-000004
Colon And Rectal Assocs of Texas	RWeygandt-C&RAT-000001-000027
Death Certificate Proof Of Authority	RWeygandt-DCPOA-000001-000004
DFW Smiles	RWeygandt-DFWS-000001-000018
Endocrine Assocs of Dallas	RWeygandt-EAD-000001-000342
Express Scripts Inc Recs	RWeygandt-ES-000001-000021
Fleshman James Jr MD	RWeygandt-JFleshamnJr-000001-000219
Heart Hosp Baylor Plano Med Recs Dept	RWeygandt-HHBP-MD-000001-000657
Hollabaugh, Eric, MD - Medical	RWeygandt-EHollabaugh-000001-000008
Lab Corp of America Med Recs Dept	RWeygandt-LabCorpA-MD-000002-000009
Legacy Heart Ctr Med Recs Dept	RWeygandt-LHC-MD-000001-000001

Med Ctr of Plano Med Recs Dept	RWeygandt-MCPlano-MD-000001-000122
Med Ctr of Plano Rad Dept	RWeygandt-MCPlano-RD-000001-000001
Med Ctr of Plano Path Dept	RWeygandt-MCPlano-PD-000001-000002
Med Clinic of North Texas PA	RWeygandt-MCNT-000001-000093
North Central Surgical Ctr	RWeygandt-NCSC-000001-000250
North Point Lab	RWeygandt-NPL-000001-000001
Plano Dermatology Assocs	RWeygant-PDA-000001-000003
Quest Diagnostics Irving	RWeygandt-QD-Irving-000001-000002
Safeway Inc Corporate Pharmacy Dept	RWeygandt-Safeway-000001-000017
Texas Health Presbyterian Hosp Dallas Patient Accts	RWeygandt-THPHD-BD-000001-000009
Texas Health Presbyterian Hosp Dallas Path Dept	RWeygandt-THPHD-PD-000001-000001
Texas Health Presbyterian Hosp Dallas Rad Dept	RWeygandt-THPHD-RD-000001-000001
Texas Oncology Pharmacy Sammons	RWeygandt-TOPS-000001-000002
Texas Oncology Plano Prestonwood Med Recs Dept	RWeygandt-TO-PP-MD-000001-000391
TMI Sports Medicine and Orthopedic Surgery - NR Cert	RWeygandt-TMISMOS-000001-000001
Verity Cancer Center	RWeygandt-VCC-000001-56
VerityPET CT	RWeygandt-VPET-CT-000001-000065
Verity PET CT Rad Dept	RWeygandt-VPET-CT-RD-000001-000087
Walgreen Company	RWeygandt-WC-000001-000006
DIOVAN NDA DOCUMENTS	
20818 Diovan Pharmacology Review Part 1 (fda.gov)	N/A
20818 Diovan Pharmacology Review Part 2 (fda.gov)	N/A
POST-MARKETING PERIODIC SAFETY REPORTS	
ANDA 077530 **Valsartan Tablets 40 mg, 80 mg, 160 mg, 320 mg**	
Teva Pharmaceuticals, 01 April 2015 – 30 June 2015	N/A
Teva Pharmaceuticals, 04 January 2016 – 03 April 2016	N/A
Teva Pharmaceuticals, 04 April 2016 – 03 July 2016	N/A
Teva Pharmaceuticals, 04 July 2016 – 03 October 2016	N/A
Teva Pharmaceuticals, 04 October 2016 – 03 January 2017	N/A
Teva Pharmaceuticals, 01 January 2017 – 31 March 2017	N/A

Teva Pharmaceuticals, 01 April 2017 – 30 June 2017	N/A
Teva Pharmaceuticals, 01 July 2017 – 30 September 2017	N/A
Teva Pharmaceuticals, 01 October 2017 – 31 December 2017	N/A
Teva Pharmaceuticals, 01 January 2018 – 31 March 2018	N/A
Teva Pharmaceuticals, 01 April 2018 – 30 June 2018	N/A
Teva Pharmaceuticals, 01 July 2018 – 30 September 2018	N/A
Teva, 01 October 2018 – 31 December 2018	N/A
ANDA 090642 **Valsartan Tablets 40 mg, 80 mg, 160 mg, 320 mg**	
Watson Laboratories, 05 January 2015 – 04 April 2015	N/A
Watson Laboratories, 05 April 2015 – 04 July 2015	N/A
Watson Laboratories, 05 July 2015 – 04 October 2015	N/A
Watson Laboratories, 05 October 2015 – 04 January 2016	N/A
Watson Laboratories, 05 January 2016 – 04 April 2016	N/A
Watson Laboratories, 05 April 2016 – 04 July 2016	N/A
Watson Laboratories, 05 July 2016 – 04 October 2016	N/A
Teva Pharmaceuticals, 05 October 2016 – 04 January 2017	N/A
Teva Pharmaceuticals, 05 January 2017 – 04 April 2017	N/A
Teva Pharmaceuticals, 05 April 2017 – 04 July 2017	N/A
Teva Pharmaceuticals, 05 July 2017 – 04 October 2017	N/A
Teva, 01 January 2018 – 31 December 2018	N/A
ANDA 091235 **Amlodipine and Valsartan Tablets 5/160 mg, 10/160 mg, 5/320 mg, 10/320 mg**	
Teva Pharmaceuticals, 01 June 2015 – 31 August 2015	N/A
Teva Pharmaceuticals, 01 September 2015 – 30 November 2015	N/A
Teva Pharmaceuticals, 01 December 2015 – 29 February 2016	N/A
Teva Pharmaceuticals, 01 March 2016 – 31 May 2016	N/A
Teva Pharmaceuticals, 01 June 2016 – 31 August 2016	N/A
Teva Pharmaceuticals, 01 September 2016 – 30 November 2016	N/A
Teva Pharmaceuticals, 01 December 2016 – 28 February 2017	N/A
Teva Pharmaceuticals, 01 March 2017 – 31 May 2017	N/A
Teva Pharmaceuticals, 01 December 2017 – 28 February 2018	N/A
Teva, 01 March 2018 – 28 February 2019	N/A
ANDA 091519 **Valsartan and Hydrochlorothiazide Tablets 80/12.5 mg, 160/12.5 mg, 160/25 mg, 320/12.5 mg, 320/25 mg**	
Watson Laboratories, 21 March 2013 – 20 June 2013	N/A
Watson Laboratories, 21 June 2013 – 20 September 2013	N/A

Watson Laboratories, 21 September 2013 – 20 December 2013	N/A
Watson Laboratories, 21 December 2013 – 20 March 2014	N/A
Watson Laboratories, 21 March 2014 – 20 June 2014	N/A
Watson Laboratories, 21 June 2014 – 20 September 2014	N/A
Watson Laboratories, 21 September 2014 – 20 December 2014	N/A
Watson Laboratories, 21 December 2014 – 20 March 2015	N/A
Watson Laboratories, 21 March 2015 – 20 June 2015	N/A
Watson Laboratories, 21 June 2015 – 20 September 2015	N/A
Watson Laboratories, 21 September 2015 – 20 December 2015	N/A
Watson Laboratories, 21 December 2015 – 20 March 2016	N/A
Teva Pharmaceuticals, 21 March 2016 – 20 March 2017	N/A
Teva Pharmaceuticals, 21 March 2017 – 20 March 2018	N/A
ANDA 200435 **Amlodipine, Valsartan and Hydrochlorothiazide Tablets 5/16012.5 mg, 10/160/12.5 mg, 5/160/25 mg, 10/160/25 mg, 10/320/25 mg**	
Teva Pharmaceuticals, 01 December 2014 – 28 February 2015	N/A
Teva Pharmaceuticals, 01 March 2015 – 31 May 2015	N/A
Teva Pharmaceuticals, 01 June 2015 – 31 August 2015	N/A
Teva Pharmaceuticals, 01 September 2015 – 30 November 2015	N/A
Teva Pharmaceuticals, 01 December 2015 – 29 February 2016	N/A
Teva Pharmaceuticals, 01 September 2016 – 30 November 2016	N/A
Teva Pharmaceuticals, 01 September 2017 – 31 August 2018	N/A
MISCELLANEOUS	
All Plaintiff Diagnosis & Treatment Report	CHODOSH0006
FDA Laboratory Analysis of Valsartan Products with Date Ranges	CHODOSH0001-CHODOSH0005
All materials cited or referenced in my expert report and curriculum vitae	N/A
All materials cited by Plaintiffs' expert witnesses - Drs. Etminan, Panigrahy, Hecht, Lagana, Madigan - in their reports and exhibits	N/A

This list includes items Plaintiffs' experts relied upon. By so doing, Defendants and this expert are not waiving any arguments or objections related to admissibility.	N/A
OTHER CORPORATE DOCUMENTS	
Aurobindo Pharma Ltd. Finished Dose Testing	APL-MDL 22875-0139456
Aurolife Finished Dose Testing	Auro-MDL 2875-0113985
Aurobindo's Initial Response to FDA's Warning Letter to Unit XI	APL-MDL 2875-0030573
Attachment 1 to Aurobindo's Initial Response to FDA's Warning Letter to Unit XI	APL-MDL 2875-0003745
Attachment 3 to Aurobindo's Initial Response to FDA's Warning Letter to Unit XI	APL-MDL 2875-0003498
Attachment 6 to Aurobindo's Initial Response to FDA's Warning Letter to Unit XI	APL-MDL 2875-0003714
Attachment 10 to Aurobindo's Initial Response to FDA's Warning Letter to Unit XI	APL-MDL 2875-0003242
Attachment 13 to Aurobindo's Initial Response to FDA's Warning Letter to Unit XI	APL-MDL 2875-0002816
Aurobindo's First Update to Response to FDA-483 Issued February 9, 2019	APL-MDL 22875-0029336
Aurobindo's Response to FDA's General Advice Letter	APL-MDL 2875-0011129
Aurobindo's Response to FDA's Warning Letter to Unit XI	APL-MDL 2875-0004189
Aurobindo's Second Update to Response to FDA-483 Issued February 9, 2019	APL-MDL 2875-0000130
Letter from Aurobindo to Lantech, dated April 1, 2019	APL-MDL 2875-2897691
Aurobindo Toxicology Health Hazard Assessment	Auro-MDL 2875-0080223
Valsartan USP and Losartan USP- API Lots Details	Auro-MDL 2875-0093561
Sartan Recalls	Auro-MDL 2875-0104586
ZHP Response to DMF Information Request Letter	PRINSTON00012480
Valsartan Detail Spreadsheet_11March2019_v32	MYLAN-MDL2875-00895544

EXHIBIT C

Testimony List – Lewis A. Chodosh, M.D., Ph.D.
1/12/2018 – 1/12/2022

1. *Ingham et al. v. Johnson & Johnson et al.* (MO) (deposition) – 4/27/2018
2. *Deisher v. Secretary of Health and Human Services* (US COFC) – 6/18/2018
3. *In re: Valsartan, Losartan, and Irbesartan Products Liability Litigation* (D. NJ) – 9/29/2021-9/30/2021